DISSERTATION

ON

FACTORS INFLUENCING PERINATAL OUTCOME OF 2ND OF THE TWIN WITH SPECIAL REFERENCE TO MODE OF DELIVERY AND CHORIONICITY.

Dissertation submitted to

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THANJAVUR MEDICAL COLLEGE,

THANJAVUR - 613 004.

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

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CERTIFICATE

This is to certify that this dissertation entitled **"FACTORS INFLUENCING PERINATAL OUTCOME OF 2ND OF THE TWIN WITH SPECIAL REFERENCE TO MODE OF DELIVERY AND CHORIONICITY**" submitted for M.D BRANCH II OBSTETRICS & GYNAECOLOGY, The Tamil nadu Dr.MGR. Medcial University, Chennai April-2013 is a bonafide work done by Dr.M.SHARADHA under my direct supervision and guidance in the DEPARTMENT OF OBSTETRICS & GYNAECOLOGY , THANJAVUR MEDICAL COLLEGE,THANJAVUR during her study period.

Dr. S.S.SWARUPARANI, M.D.,D.G.O.,
Professor and Head of the Department,
Department of Obstetrics & Gynaecology,
Thanjavur Medical College.
Thanjavur - 613 004.

Prof. Dr.C.GUNASEKARAN M.D.DCH.,
The Dean I/C,
Thanjavur Medical College.
Thanjavur - 613 004.

Thanjavur Medical College

THANJAVUR, TAMILNADU, INDIA-613004

(Affiliated to the T.N Dr.MGR Medical University, Chennai)

ETHICAL COMMITTEE

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Name of the Candidate	:	Dr.M.SHARADHA
Course	:	M.D.(O.G)
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DECLARATION

I, **Dr.M.SHARADHA**, solemnly declare that the dissertation titled **"FACTORS INFLUENCING PERINATAL OUTCOME OF 2ND OF THE TWIN WITH SPECIAL REFERENCE TO MODE OF DELIVERY AND CHORIONICITY"** is a bonafide work done by me at Department of Obstetrics & Gynaecology, Thanjavur Medical College, Thanjavur under the guidance and supervision of my beloved **Prof.Dr.S.SWARUPARANI, M.D.,D.G.O.,**

This dissertation is submitted to The Tamilnadu Dr. M.G.R Medical University towards partial fulfilment of requirement for the award of **M.D. degree (Branch -II) in Obstetrics and Gynaecology** degree examination to be held in April 2013.

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Date: - 12 - 2012 M.D Post graduate

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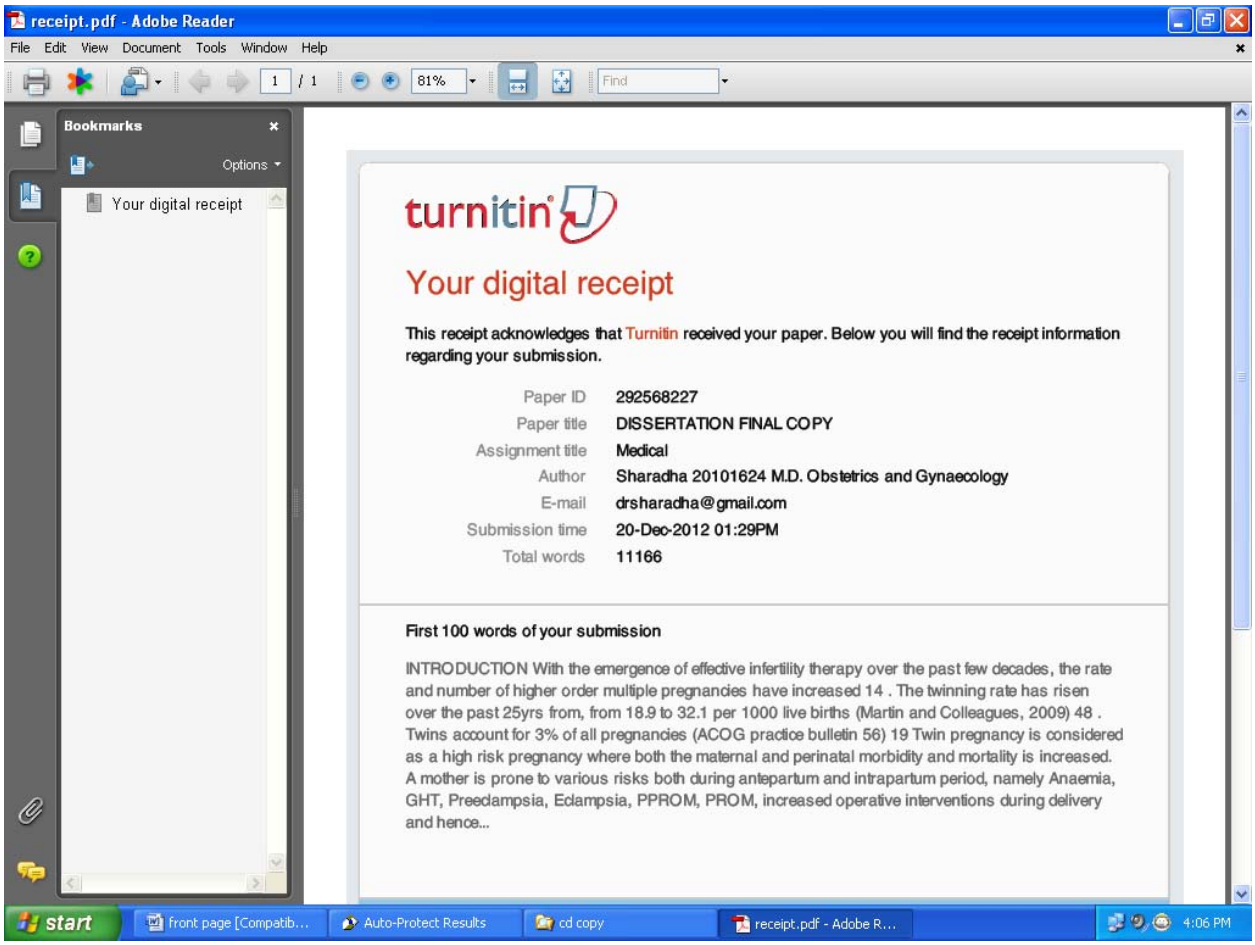
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Author Sharadha 20101624 M.D. Obstetrics and Gynaecology
E-mail drsharadha@gmail.com
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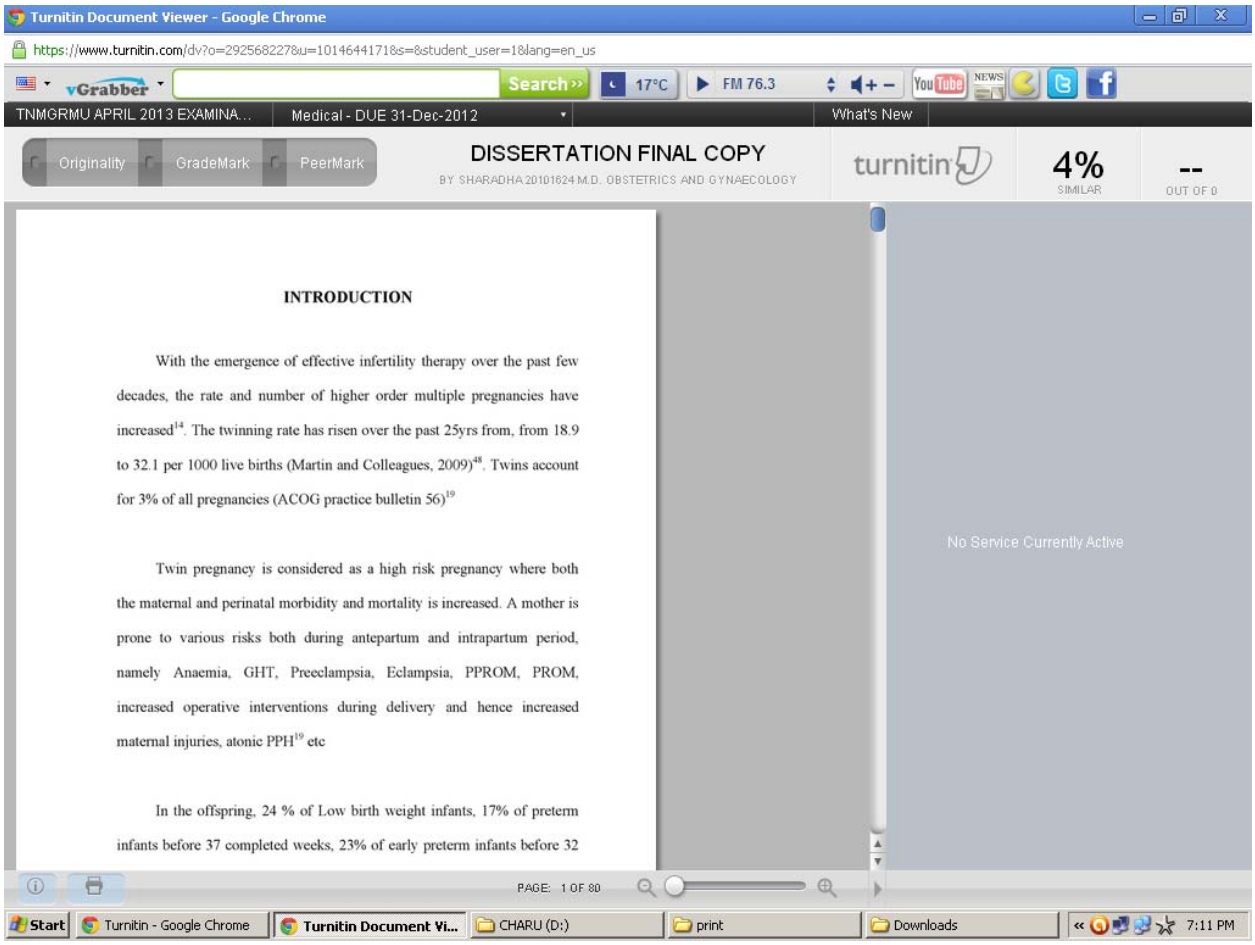
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FACTORS INFLUENCING PERINATAL OUTCOME OF 2ND TWIN WITH SPECIAL REFERENCE TO MODE OF DELIVERY AND CHORIONICITY.

Absract:

Introduction: Multiple gestation is often considered a novelty with pleasant images of identical children dressed alike. They account for 3% of all births and considered as one of the high risk pregnancy.

Aims & objectives: To study the natural course of twin pregnancies, and to compare the perinatal morbidity of the second twin with the first twin and the effect on chorionicity.

Materials and methods: This is a Prospective study from June 2011 to July 2012 , of all twin pregnancies with gestational age > 28 weeks, excluding IUD of 2nd twin. All cases were analysed based on chorionicity, looked for discordancy, anomalies, fetal well being and decided on the apt period of termination for each kind of twin. The perinatal outcome were analysed based on APGAR scores and duration of NICU stay and their discharge status.

R**esults**: Incidence of twins during the study period was 16.3/1000,out of them 200 cases were taken and analysed. Majority of the women fell in the age group of 21-25yrs (46.5%), more common in primi (53%). Based on chorionicity, there were 57% of DCDA, 39.5% of MCDA, 3.5% of MCMA twins. Prematurity is the most important cause of morbidity in twins,with 53.5% of cases delivered before 37 weeks resulting in 85.9% of perinatal deaths. As the time interval between delivery of the twins increases, the mortality is high. <10 minutes -15%,11-20mins

-17%,>20mins-22%.Discordancy is high with MC twins (20) than DC twins(15),loss percentage is 22.2% in 1st twin and 46.17% in 2nd twin and the mortality for the discordant 2nd twin in MC pregnancy is still higher.APGAR scores were poor for the 2nd twin.

Conclusion: In our study we observed that the perinatal mortality was high for the 2nd twin (160/1000 Vs 125/1000).The perinatal loss was high with MC twins than in DC twins (63.1% vs 36.8%).We observed that the perinatal outcome was not influenced by the mode of delivery.

INTRODUCTION

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Twin pregnancy is considered as a high risk pregnancy where both the maternal and perinatal morbidity and mortality is increased. A mother is prone to various risks both during antepartum and intrapartum period, namely Anaemia, GHT, Preeclampsia, Eclampsia, PPROM, PROM, increased operative interventions during delivery and hence increased maternal injuries, atonic PPH[19] etc

In the offspring, 24 % of Low birth weight infants, 17% of preterm infants before 37 completed weeks, 23% of early preterm infants before 32 completed weeks, (ACOG 2004) occur in twin pregnancies, added on to this are IUGR babies[19].

Whenever a woman is diagnosed to have twin pregnancy, the type of placenta is determined followed by adequate counseling of the woman regarding extra calorie intake throughout pregnancy, regular antenatal visits and ultrasound to monitor the fetal growth, their wellbeing, picking up anomalies, IUGR and discordancy at the earlier onset, and plan accordingly.

The Obstetrician should anticipate and aim to prevent preterm birth, by advising the woman to take adequate bed rest, Antenatal steroids, and should plan for the best mode of delivery in each case[35]. With a Multidisciplinary approach involving skilled obstetrician, Anaesthesiologists, Neonatologists each women bearing twin pregnancies can have a successful pregnancy outcome with less fetal and maternal morbidity[53].

REVIEW OF LITERATURE

Twins are 2 offsprings from a single pregnancy. There are 2 types of twinning, dizygotic and monozygotic.

Non-Identical/dizygotic/fraternal twins- Born from fertilization of 2 separate ova, they cannot be considered as true twins, each of them have a different genetic growth potential since they are a product of maturation and fertilization of 2 separate ova during a single cycle[14].

Identical/monozygotic twins - Born from fertilization of a single ovum, that splits into two. This process of splitting itself is a teratogenic event resulting in more discordant malformations in monozygotic twins, involving asymmetrical organs like heart. They may not be identical, as the splitting may not result in equal sharing of protoplasmic material, also they can even be discordant for genetic mutations due to postzygotic mutation, or may have varied expression of same genetic disease, (Glinianaia et al 2008)[67].

Sophisticated genetic testing is needed to determine the zygosity.

The Chorionicity in monozygotic twins is based on time of splitting of the fertilized egg. Occurrence of splitting in three days of fertilization,

results in Dichorionic twins (DC). Splitting after 3rd day results in monochorionic (MC) twins[14].

Cell stage	Time of cleavage	Type of placenta
Morula	1-3 days	Dichorionic diamniotic (30%)
Blastocyst	4-8days	Monochorionic diamniotic (65%)
Implanted blastocyst	8-13days	Monochorionic monoamniotic (5%)
Formed embryonic disc	>13days	Conjoined twins

The etiology of monozygotic twins are not known and its frequency is constant in terms of 1 in 250 births, forming 1/3rd of twin pregnancies, whereas the incidence of dizygosity establishes 2/3rd of twin pregnancies and its occurrence is influenced by various factors[14].

FACTORS INFLUENCING DIZYGOTIC TWINNING:

1. Race:

Frequency of twinning varies in different races and groups of various ethnicity, it is more prevalent in the rural community called Yoruba tribe of Nigeria where Morley and Knox (published in BJOG vol., 67)[54], found twins occurring once in every 20 births. These differences in different ethnic

communities may be due to FSH level variations since FSH levels are raised in dizygotic twin pregnancies. A study by Ibrahim I et al[42] in Niger delta (2007 – 2010) quoted the incidence to be high of 30.6/1000 live births in Nigeria.

2. Heredity:

A positive family history especially on the maternal side has more significance. Woman who are actually dizygotic twins themselves, gave birth to twins at an increased frequency than a positive history on the paternal side, the incidence of the former being 1 in 58 births, versus 1 in 116 pregnancies of the latter (white & wyshak in 1964) [18]

3. Maternal age and parity:

As age advances, with maximal FSH stimulation, multiple follicles develop, increasing the twinning rate. The peak age is 37yrs (Beemsterboer and co-workers 2006) [46].A fall thereafter is due to the depletion of the follicles. Similarly with increasing parity twinning incidence increases, frequency of multiple pregnancies in 1stconception was 1.3% versus 2.7% in 4th pregnancy (pettersson & associates 1976) [55]. Azubuike(1982) [45] in Nigeria showed that as parity advances, the incidence rose from 2% in primiparous woman to 6.6% in a woman pregnant for 6th time or more !

4. Nutritional factors:

Well nourished woman with high BMI are more prone to have twins. Haggarty and associates (2006) [47] reported that increased folate consumption increased twinning rates.

5. Pituitary gonadotropin:

A sudden release of FSH, one month after stoppage of oral contraceptive pills, in the 1st spontaneous cycle, results in increased fecundity and hence increased incidence of twinning. This however does not happen in the subsequent months. (Rothman 1977) [49]

6. Infertility therapy

Ovulation induced cycles with clomiphene citrate, FSH plus HCG, gonadotropin therapy increases twinning rates. Schenker and cowokers in 1981[50] reported 16 to 40% multiple gestation with gonadotropin therapy, of which 75% were twins, similar results were given by Tuppin and coworkers in 1993[56] in France. Risk of multiple gestations associated with these therapies may be as high as 25% (ACOG 56) [19]

7. Assisted reproductive technology:

This technology increases the probability of conception. The probability of multiple pregnancy increases with ART techniques. In 2005 out of 1% of

infants born through ART 17% were multiple births in United States (Wright & colleagues 2008) [52] .The risk of twins and multiple fetuses depends on the number of embryos transferred.

How to determine chorionicity?

Since the chorionicity has many implications in antepartum and intrapartum management, it is the chorionicity and not the zygosity that is important. Dichorionic, whether Monozygotic / Dizygotic develops as 2 distinct individual organs and hence are not at increased risk. Whereas Monochorionic twins are at increased risk due to vascular anastamoses between the 2 circulations. In 10-15% cases of MC twins, Twin to Twin transfusion syndrome (TTTS) can develop having a poor perinatal outcome[27].

Chorionicity can be determined either sonographically or by post delivery examination of placenta. However sonographic evaluation has 96% accuracy[68]. Presence of 2 separate placentas with a dividing membrane of 2mm thickness or more indicates dichorionic pregnancy. Here, tissue from the anterior placenta will be found to be extending down, between the amnion layers. This sign namely **twin peak sign** or **lambda sign** is specific for dichorionic twinning. In DCDA (Dichorionic Diamniotic) twins, the 2 placentas may be fused or separated. Absence of the above sign and the presence of a thin dividing membrane <2mm thick, is called the “T” sign. Here the twins are separated by a

thin diving membrane created by the amnion of each twin and the magnification of the membrane reveals only 2 layers. This is the feature of Monochorionic Diamniotic twins (MCDA). It is accurate in 82% of Monochorionic and 95% of Dichorionic pregnancies when done before 14 weeks of pregnancy[40].

Counting the number of layers in the dividing membrane can also be used for finding out the Chorionicity; it is more accurate with a high frequency probe of the range 7.5-20 MHz. With this technique the predictive value of chorionicity is almost 100%[58].

For determination of Monoamniotic placentation, the sonographer must be 100% sure about the presence /absence of the dividing membrane. With the bad prognostic implications of MCMA pregnancies, atleast 3 different ultra sound examinations has to be done before concluding that it is MCMA (Monochorionic monoamniotic) placenta. Visualisation of 1 yolk sac with 2 fetal poles before 8wks confirms MCMA placenta[63]. In majority of cases, Cord entanglement can be imaged by using Color Doppler Ultrasound in the 1st trimester of pregnancy by 14 weeks[40].

Placental examination after delivery includes looking for chorion intervening between 2 amnions .With 2 amnions and 2 chorions it is dichorionic

twinning, with dizygosity being more common. With no chorion in between the juxtaposed amnions, it is monozygotic pregnancy.

Neonates of different sex and blood groups confirm dizygosity, but demonstrating the same sex/blood group does not confirm monozygosity[57]. Definitive diagnosis is possible with DNA finger printing, and they are indicated only if there is a pressing medical indication.

PREGNANCY OUTCOME:

MATERNAL COMPLICATIONS	FETAL COMPLICATIONS	PLACENTAL COMPLICATIONS
Abortions	Discordant growth	Abruptioplacenta
Hyperemesis gravidarum	Congenital anomalies	Cord accidents
Anaemia	IUGR, prematurity, LBW	Cord entanglement, cord prolapse
Gestational hypertension & diabetes	Fetal demise of one or more twins	PPROM
Preeclampsia	Malpresentations	Postpartum bleeding
Preterm labour	TTTS(Twin to twin transfusion syndrome)	Placenta previa
Operative deliveries and maternal injuries	TRAP(Twin reversed arterial perfusion)	

Perinatal mortality varies with the type of placentation and birth order, A special reference to increased morbidity and mortality of the 2nd twin (276/1000

vs 138.5/1000 births of 1st twin) pandole et al (Mumbai 2003) [8] is due to increased risk like low birth weight, IUGR, discordancy, Malposition, assisted delivery, placental separation, cord prolapse, uterine atony, cervical spasm, anaesthetic risk, long intertwin delivery interval. Also the safe mode of delivery of the 2nd twin in non vertex presentations is a subject of controversy among the obstetricians [30].

Prematurity is the most important cause of death in twin pregnancy[24]. The serious consequences of prematurity are- hypothermia, PDA, respiratory difficulties , hypoglycemia , NEC , sepsis , retinopathy of prematurity ,cerebral palsy, multicystic encephalomalacia, porencephaly[27].

UNIQUE COMPLICATIONS IN TWINS:

1. VANISHING TWIN:

In established cases of twin pregnancy by 1st trimester, one twin may spontaneously abort/vanish before the 2nd trimester. The pregnancy continues as singletone with no complications on the survivor twin (Landy HJ et al 1986 [43]).It occurs in 20 to 60% of cases of spontaneous twin conception. This condition may result in elevated serum maternal and amniotic fluid serum alpha – fetoprotein level, and a positive amniotic fluid acetyl choline esterase assay. (Winsor and associates, 1987)[51]. This may therefore interfere with the screening of mother for down syndrome or neural tube defects, hence in suspected cases,

it is necessary to establish the diagnosis of vanishing twin before we screen for down syndrome in the early trimester.

2. FETUS PAPYRACEUS:

Fetal death usually occurs around 16 to 20 weeks of pregnancy. Body fluids are absorbed, and the fetus in flattened by the continued growth of the survivor twin. It is more common in monozygotic twins. Fetus papyraceus occurs in 1 (0.54%) of 184 of twin pregnancies (Livnat et al 1978)[44]

3. DISCORDANT GROWTH:

It means a difference of >20% in the birth weight of both twins, graded as, Grade I – 15 to 25% , Grade II > 25% , based on birth order of the discordant twin they are called as discordant 1st or 2nd twin, the prognosis for the 2nd twin is worse when it is discordant. The causes for discordancy may be genetic anomalies, unequal placenta and TTT syndrome.

VARIOUS ULTRASOUND CRITERIA USED TO DIAGNOSE DISCORDANCY

1. Difference of Biparietal diameter of 6mm or more.
2. Head circumference difference of > 5%

3. Difference of Abdominal circumference of 20mm or more

4. Difference of 15-20% in estimated fetal weight has been in use recently.

Discordancy due to unequal placental mass is more common with dichorionic twins. It can be identified after 24 weeks[27].These babies are asymmetrically discordant. Uterine & umbilical artery doppler shows evidence of increased impedence in flow. In severe cases, evidence of Fetal heart rate changes and fetal distress and acidosis, and the fetus may succumb. These babies are monitored with weekly Non stress tests, Doppler studies and Biophysical profile.

If the cause is due to genetic syndromes, they can be identified by 16-20 weeks, more commonly seen with monochorionic twins. They result in symmetric discordancy. It is not associated with any Doppler changes unless there is a placental insufficiency. Management depends on the nature of the genetic disorder & its corrective possibilities & future survival of the babies.

4. TWIN TO TWIN TRANSFUSION SYNDROME:

This phenomenon is unique to Monochorionic placentas where, blood from the donor twin is being transfused to the recipient twin, resulting in anaemia and severe growth restriction of the donor twin and polycythemia with circulatory overload with hydrops in the recipient twin[27].

The Mechanism in TTTS is unidirectional deep A-V anastamoses, resulting in unbalanced net flow between the fetuses. If it is associated with compensatory A-A anastamosis, the prognosis will be better as seen in MCMA twins, hence TTTS is more common with MCDA twins (10-15%) than with MCMA twins. This may also be associated with unequal placental mass resulting in discordancy making diagnosis of this condition difficult[27]

Acute TTTS occurs in the intrapartum period due to delayed clamping of umbilical cord following delivery of 1st twin resulting in retrograde flow of 2nd twin's blood into 1st twin. Even pronloged intertwin delivery interval time between both twins can cause loss of blood from 2nd twin into 1st twin's placenta[27]. Hence the delivery interval time should not be prolonged more than 15-20 minutes in Monochorionic pregnancies. It is identified by difference in haemoglobin (Hb) concentration of at least 5 g/dl between both the twins at birth and the presence of unbalanced placental vascular anastomoses being identified by the pathologist. This has been observed by Hack et al[27], where he studied a course of 617 MCDA pregnancies and found a relation between mode of delivery and Hb-level after birth. It appears that after vaginal delivery, second-born twins had a higher Hb compared to their co-twin.

Diagnostic criterias in the Antenatal period are:

- Finding a single placenta in ultrasound
- Same sex twins
- Presence of discordancy
- Presence of significant difference in the liquor volume of each twin. Oligohydramnios where deepest vertical pocket ≤2 cm in the sac of one twin and polyhydramnios (deepest vertical pocket ≥8 cm before 20 weeks of gestation or ≥10 cm after 20 weeks of gestation) [27] in the sac of the other twin. In appropriate cases, these women can be referred for laser coagulation of the anastomotic vessels.
- Hydrops in any of the twins or both
- Umbilical cords should have different sizes
- Hemoglobin difference > 5g/dl
- Umbilical artery Doppler changes

"STUCK TWIN"-poly – oli syndrome[61] is to mean the sonographic appearance of the donor twin in extreme forms of TTTS, with literally no amniotic fluid around the donor twin and it appears to be stuck to the uterine wall. The mortality, if not treated is 80%.

Though it presents between 18 and 26 weeks, rate of Survival for those cases diagnosed before 28 weeks widely varies from 7-75% (berghella and Kaufmann, 2001)[61]

Incidences of CNS complications like periventricular leukomalacia and intra ventricular hemorrhage are greater in these twins. Cardio vascular complications like cardiac hypertrophy with tricuspid regurgitation and renal failure etc can also occur.

STAGING OF TTTS BY QUINTERO[62]:

Stage 1- Poly/oligoamnios with presence of fetal bladder in donor twin

Stage 2- Stage 1 with absence of bladder in donor twin, with normal Doppler studies

Stage3- Stage 2 with abnormal Doppler changes like absent or reversed diastolic flow in umbilical artery/ductus venosus in donor twin or pulsatile flow in umbilical vein of recipient twin

Stage 4: Emergence of fetal hydrops

Stage5: Demise of 1 or both twins.

Presence of A-A anastamoses identified with color Doppler by 18 weeks is advantageous, with a survival rate of 83% .Its presence, subdivides stage 3 as

3A, & its absence being associated with poor survival rates is graded as stage 3B.

Several therapeutic modalities namely, laser ablation of vascular anastamoses, serial amnioreduction, amniotic septostomy and selective feticide have been tried of which laser photocoagulation has been found to be highly effective. The survival rate for donor fetus is 70.5% and recipient is 72% (Huber et al 2006) [4]

5. FETAL DEMISE OF ONE TWIN

More common with monochorionic twins, 2.6% versus 1.1% in dichorionic twins, discordancy is an important risk factor; even 20% of discordancy has an increased risk in monochorionic twins. There is six fold greater risk of death in the surviving twin following IUD of 1 twin[60].

In cases of single fetal death, in MC twins, vascular anastomoses in the shared MC placenta, can cause significant shunting of blood between both fetal circulations and put the MC co-twin at higher risk of perinatal mortality and morbidity than in DC twins (BJOG 2006).[71] Consequences to the surviving co-twin can be profound, including co-twin death, survival with cerebral impairment or preterm delivery with its sequelae. Even after excluding

antenatally diagnosed TTTS, mortality is still higher in MC twins than in DC twins.

The prognosis of the surviving twin depends on the gestational age at the time of demise, the chorionicity and the length of time between the demise and the delivery of the surviving twin. A fetal demise before 14weeks has no increased risk on the survivor, but the prognosis after the 1st trimester purely depends on the chorionicity[59].

Death of one fetus causes massive transfusion of blood from the survivor to the dead twin, resulting in anemia which can be detected by middle cerebral artery Doppler. This causes neurological, cardiac, renal morbidity due to ischemia of the surviving twin. The typical neurological lesion is multicystic encephalomalacia[41]. In monochorionic twins the mortality of the other twin rises to 50% and if at all the 2nd twin survives, the neurological morbidity is around 30%.

In a study by Nayak et al, 2003[7] in Mumbai, intrauterine demise of 1 fetus by 22weeks and pregnancy was closely monitored and conserved with the result of a live birth of the surviving fetus by 36weeks.

Demise at a later gestational age has a greater risk for the other twin and the mother. Maternal complication in terms of DIVC is a rare phenomenon, and it occurs usually >3 weeks after the demise and has a spontaneous resolution.

Intervention is not needed at this time since severe bleeding from the survivor twin might have occurred within few minutes after the fetal demise (karsidag et al., 2005)[66], and since it's not possible to know how many hours or days have elapsed since the death and the neurological insult might have happened before we could intervene. The adverse effect of delivering the baby preterm should also be taken into account. If MCA Doppler reveals severe anemia of the surviving twin then intrauterine transfusions can be tried to avoid further ischemic damage. MRI of fetal brain 2-4weeks following demise can identify ischemic lesions and redefine prognosis of fetus[14].

6. CONGENITAL ABNORMALITIES:

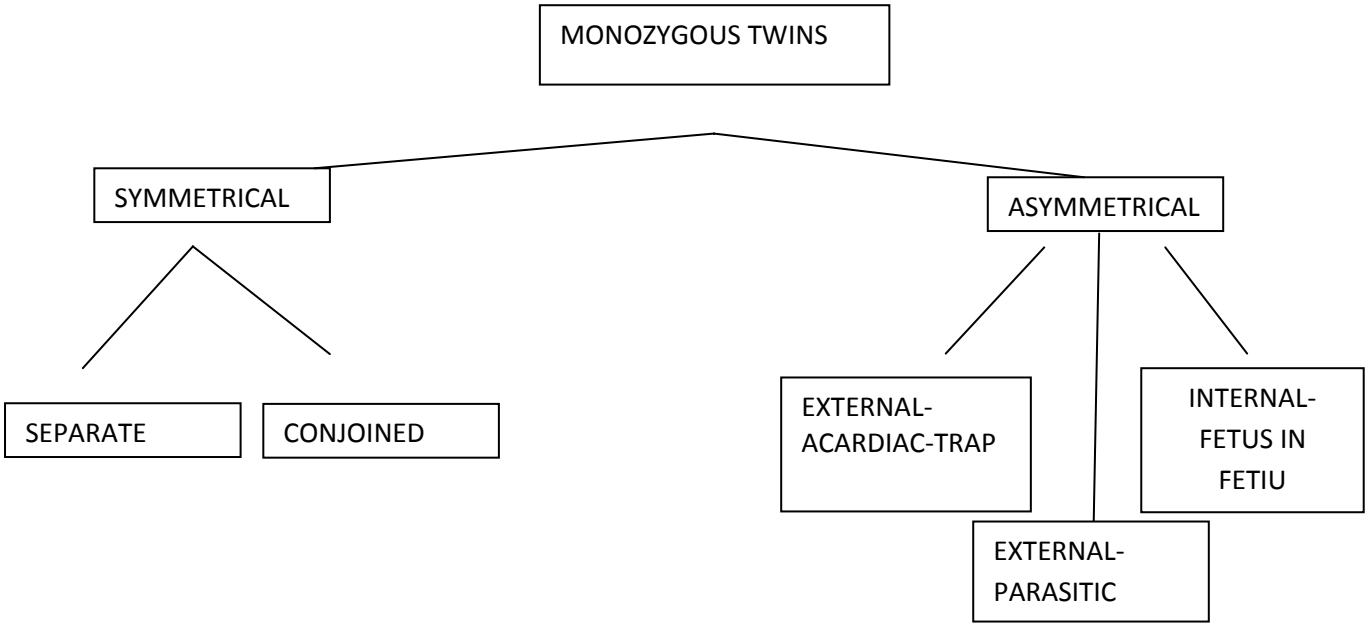
Incidences of congenital malformations are high in twin pregnancies compared with singleton. This is consistent in various studies, a study by Cameron et al 1983[2] found an incidence of 3.7 and 2.5% in monozygotic and dizygotic twins respectively. Monochorionic monoamniotic twins have the highest incidence of risk around 26%. Common abnormalities include neural

tube defects (33%), abdominal wall and genitourinary system abnormalities (33%), cardiac (14%), isolated limb abnormalities (8%).

Malformations in monozygotic twins are lethal and multiple, and those in dizygotic twins are minor.

Anomalies specific to twins are fetal acardia and conjoined twins.

MONOZYGOTIC TWINS:



MONOAMNIOTIC TWINS:

They are rare, occurring 1 in 12,500 births; perinatal mortality is high around 50%. Congenital anomalies, preterm births, low birth weight, discordancy are the complications. TTTS is less due to presence of compensatory superficial A-A anastamoses. The most important complication

causing significant morbidity is cord entanglement. They can be detected antenatally by 14 weeks with ultrasound. Once identified, they are subjected to Doppler studies; the key is to look for diastolic notching in the umbilical artery or high velocity blood flow in the umbilical vein.

Presence of variable deceleration in CTG tracing, requires frequent fetal assessment, sonography every two weeks and termination of pregnancy by cesarean section by 34 weeks. During the antenatal period women are admitted by 28 weeks and monitoring of fetal hearts for a minimum of 2 hours per day have improved the outcome of these pregnancies (De Falco and associates in 2006)[63]. Reduction of amniotic fluid volume by using indomethacin or sulindac will limit the fetal movements thereby avoiding cord entanglement. Though sulindac causes constriction of ductus arteriosus, such an effect of it is not pronounced (sawdy et al., 2003).[11]

CONJOINED TWINS

They result from incomplete splitting of an embryo into 2 separate twins (kaufman 2004)[64].They are called after Chang and Eng Bunker of Siam (Thailand) as Siamese twins. There are various characteristic forms of them based on joining of the twins at either pole. The most common form is paraphagus (spencer 2001)[12].

They can be identified by midpregnancy ultrasonography, time enough for the parents to make a decision. A careful targeted evaluation of the points of connection and the involvement of various organs is needed before counseling a patient. Better anatomical information can be provided by MRI. If essential organs have not been shared, surgical separation might be successful.

A combined effort from the pediatric surgeon will assist the parents in taking a decision. These twins may have discordant structural anomalies complicating the decision of continuing pregnancy.

A viable twin is to be delivered by LSCS to avoid traumatic delivery. In case of termination in earlier weeks vaginal delivery can be allowed since the union is most often pliable.

EXTERNAL PARASITIC TWINS:

A grossly defective fetus or only few fetal parts remain attached to a normal twin. They are the result of remnants of a demised twin gaining vascularity from the normal twin. They are merely supernumerary limbs with some viscera, with no functioning vital organs (spencer, 2001)[12].

FETUS IN FETU:

This represents an enfolded embryo acting as a parasite inside its normal twin, supported by large parasitic vessels. Due to the arrest of their normal growth in 1st trimester, these fetiform masses are represented by vertebral and axial bones with loss of normal spatial arrangement of organs, and hence lack vital organs like heart and brain(spencer,2000a)[69].

ACARDIAC TWIN:

Rare anomaly seen in 1 per 35000 live births. The Acardiac twin lacks heart and placenta, hence derives its blood supply through A-A anastamoses from donor twin. The deoxygenated blood (TWIN REVERSED ARTERIAL PERFUSION) supplies mainly the lower extremities through the umbilical and iliac arteries, resulting in a poor perfusion to the upper part of the body resulting in non development of heart and upper extremities. The circulatory overload on the healthy twin's heart results in high output failure raising its mortality to 50 %.(Faye –Petersen et al 2006)[70]

THREE TYPES-

- Acardiac-acephalus-thoracic organs & fetal head are absent
- Acardiac-amorphous-unrecognised fetal parts in a mass of tissue
- Acardiac-myelacephalus-head with one or more extremities develop

There is confusion in the diagnosis due to their resemblance with anencephalic fetus and they are mistaken for IUD due to their lacking of heart.

Management of this condition is complex, expectant management results in spontaneous stoppage of blood flow from the normal twin and its survival has been observed (Sullivan et al., 2003) [13]

Interruption of the A-A anastamoses by endoscopic laser photocoagulation, selective removal of acardiac twin, cardiotonics to mother or fetus etc has been tried.

INTERLOCKING OF TWINS:

When the babies present as breech – cephalic this complication is to be anticipated, where the twin 2's head is interposed between the body and the head of the 1st twin. This results in abnormal labor pattern- an arrest disorder. In such cases best is to proceed with LSCS, and the mortality for the 1st twin is 100%. Perinatal mortality on the whole is 62-84%

Locked twins have been reported in 1:817 twins, misra and tripathy in 2002[6] from cuttack and patil and rita[9] in the same year from hubli.

ANTEPARTUM CARE:

Every patient is to be scheduled for Antenatal visits every 2 weeks, USG every 3 – 4 weeks, vaginal and bladder infections need to be treated promptly. Women should be advised about 14 hours of rest per day. Administer Antenatal steroids when needed. Each woman should take 300kcal extra than in a singletone pregnancy. This also includes 60mg/day of iron and folic acid 1mg/day.

RCOG GUIDELINES: [53]

DICHORIONIC TWINS:

- Multidisciplinary approach.
- USG by 10-13 weeks for viability, chorionicity and NT scan for Aneuploidy.
- Detailed anomaly scans by 20-22 weeks.
- Serial scans to monitor the growth of the fetuses by 24, 28, 32 thereafter 2 weekly or 4 weekly.
- Monitoring BP and urine for albuminuria every 4 weekly starting from 20 weeks to 28 weeks, 2weekly thereafter.

- Discuss about the mode of delivery by 34-36 weeks.
- Plan to deliver electively by 37-38 completed weeks.
- Postnatal care and advice regarding breast feeding and contraception.

MONOCHORIONIC TWINS:

Few exceptions on comparison with DC twins are:

- Screen for TTTS along with Aneuploidy by 10-13 weeks scan.
- Ultrasound by 16weeks and then 2weekly thereafter to check for development of TTTS and discordancy.
- Anomaly scan by 20-22 weeks should include fetal ECHO too.
- Growth monitoring scans every 2 weekly until delivery.
- Discuss about the mode of delivery by 32-34 weeks.
- Plan to deliver electively by 36-37 completed weeks in uncomplicated cases.

TIMING OF DELIVERY:

Though 50% of the twin pregnancies end in preterm labor[25], all uncomplicated DCDA pregnancies should be terminated by 38 completed weeks and those with complications should be induced before 38 weeks, and it

is not necessary to do tests of fetal lung maturity. The risk of intrauterine fetal death is high than the risk of neonatal death if these pregnancies are allowed to continue till 40 completed weeks. All MCDA pregnancies terminated by 36-37weeks and MCMA pregnancies by 34 weeks, mortality being 10% if allowed to continue versus neonatal death of 1%.

The optimal mode of delivery should be decided before labour or in early labour, and the route of delivery is based on the fetal presentation, amnionicity and additional maternal & fetal risk factors.

In case of cephalic - cephalic twins vaginal delivery is the best option[19], controversy arises in non-vertex 2nd twin.

Gathered from various reports, the morbidity and mortality of the 2nd non vertex twin weighing >1500g is not increased, irrespective of the route of delivery. Caesarean section should be performed only in case of poor progression of labor, or fetal indications like fetal distress, hyperextended head of 2nd twin, or if 2nd twin is much larger than first, cord prolapse (blickstein I .1987)[65.] . Similar observations have been put forward by Greig P.1992, chevernak F. (1984)[22] non-vertex twins can be safely delivered vaginally if they weigh >1500g.

After delivery of 1st twin external version of 2nd twin is successful in 40-50% of cases, vaginal breech delivery can be done in 96% of cases. Main problem with 2nd twin presenting as breech is increased chance of cord prolapse.

AIMS AND OBJECTIVES

1. To study the natural course of twin pregnancies.
2. To evaluate the maternal and perinatal complications in twin pregnancies.
3. To study the perinatal morbidity and mortality of both the twins in a twin gestation.
4. To compare the perinatal morbidity of the second twin with the first twin.
5. Outcome of the babies with respect to chorionicity.
6. To study the relationship of several of these factors to perinatal mortality,
    - Chorionicity
    - Gestational age of delivery
    - Influence of maternal high risk factors
    - Weight discordancy
    - Delivery Time interval between the fetuses
    - Presentation of the babies
    - Mode of delivery of the babies
    - Birth weight of each twin
    - 5minute APGAR of the babies
7. To study the various causes of perinatal mobidity and causes of death in the neonate

MATERIALS AND METHODS

The clinical material for this study was taken from the Department of Obstetrics and Gynaecology, Raja Mirasudar hospital, Thanjavur Medical College. The study period was from June 2011 – July 2012. During this period 14300 patients were admitted for delivery. Out of them, there were 234 twin deliveries. 200 cases were taken for the study after satisfying the exclusion criteria.

Inclusion Criteria:

All women with twin pregnancies of >28 weeks of gestation.

Exclusion Criteria:

1. Cases with gestational age < 28 weeks
2. Those pregnancies with IUD of 2nd twin
3. Women with uncertain last menstrual periods.

On routine antenatal visits a detailed history was taken from each woman pertaining to twin pregnancy, about their LMP, past obstetric history, past history of twin pregnancy, family history of twin pregnancy, history of ovulation induction or other ART measures. Whether USG was done, at what weeks of GA was USG done, whether told about any anomalies, discordant growth.

Detailed history in each trimester regarding any complaints in the antenatal period like, excessive vomiting, features suggestive of GHT, anaemia, urinary tract infections. History of adequate calorie intake during each trimester, history of hours of bed rest per day, intake of tocolytics , or Antenatal steroids. Routine examination consisted of looking for features of anaemia, GHT, position & presentation of fetuses, fetal parts and heart sounds.USG was done to confirm the diagnosis, presentation of the fetuses, looked for anomalies, birth weight discordancy etc. Routine investigations were done.

In patients admitted with labour pains, Gestational age in weeks was calculated from day one of the last menstrual period. Presence of complications were looked for, like, PROM, PPROM, preterm labour, abruption, presentation of fetuses, mode of delivery, time interval, APGAR scores, complications of 3^{rd} stage labour in the mother were noted. Placenta was examined postnatally and the chorionicity noted.

Perinatal outcome was measured in terms of number of babies admitted to the neonatal intensive care unit, the number days of admission into NICU, and the final outcome of the babies, in terms of whether discharged in good condition or expired during the neonatal period. Neonatal morbidity was further defined based on the causes like, respiratory distress syndrome (RDS), septicemia, intra uterine growth restriction (IUGR), neonatal hyperbilirubinemia

(NNH), patent ductus arteriosus (PDA), hypoglycemia, anomalous baby, neonatal seizures (NNS).

Causes of death were termed as due to Birth Asphyxia, Sepsis, Cord prolapse, Prematurity & its complications, Anomalous baby, Fetal growth restriction, neonatal seizures, intra uterine death.

The perinatal loss was defined as Intrauterine death or Neonatal death (≤28 days of birth) having a birth weight of >1 kg. Still births were also included in perinatal mortality. Stillbirth was Intra uterine death of a fetus weighing >1 kg and/or ≥28 weeks of gestation. Stillbirths were divided as ante partum deaths, where the fetuses had died before the start of labour, and intrapartum fetal deaths, where the fetuses had been alive at the onset of labour.

Perinatal morbidity was defined as 5-minute Apgarscore <7. Preterm delivery was defined as those delivered before 37 weeks of gestation and very preterm delivery as delivery was defined as those delivered before 32 weeks. Low birth weight was defined as birth weight less than 2.5kgs.Asphyxia was defined as a 5-minute Apgar score <5.

RESULTS AND ANALYSIS

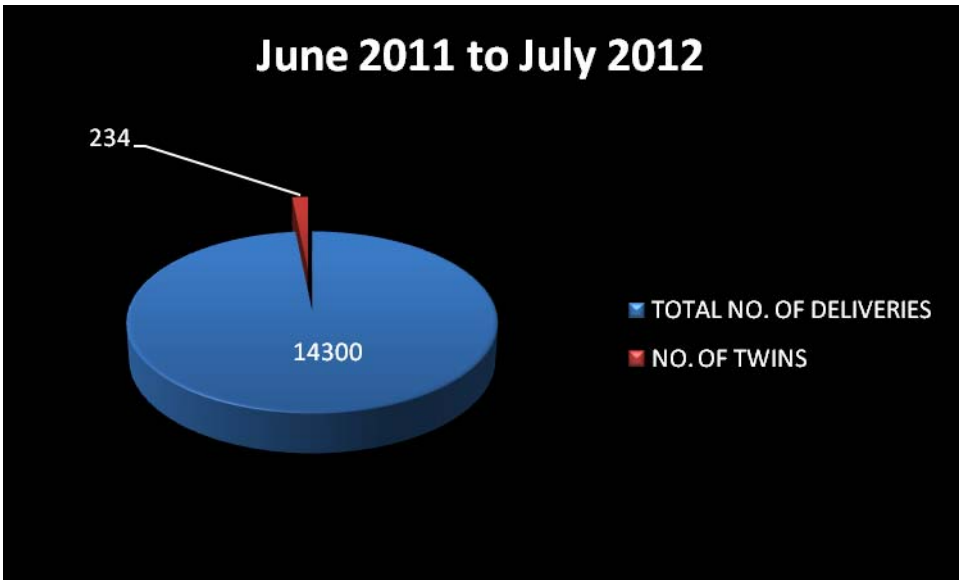
The study about twin pregnancies commenced from June 2011 to July 2012.The study material consisted of 234 cases.200 cases were taken for the study after satisfying the exclusion criteria. The general incidence of twins in total live births and their incidence in relation to various maternal factors like Age, Parity, Conception arising out of ovulation induction and spontaneous conceptions, Incidence in relation to hereditary factors, maternal complications due to twin pregnancies were analysed. Finally the perinatal outcome of the 2nd of the twin with special reference to its mode of delivery and chorionicity were studied and analysed.

1. INCIDENCE OF TWIN PREGNANCY

TABLE 1

YEAR	TOTAL NO. OF DELIVERIES	NO. OF TWINS	INCIDENCE
June 2010 to July 2011	15565	210	13.49/1000
June 2011 to July 2012	14300	234	16.3/1000

FIGURE-1



The incidence in the study period was 16.3/1000 live births, with the actual number being 234.

TABLE-2

S.NO	BOOKING STATUS	NUMBER
1.	BOOKED	152
2.	UNBOOKED	48

Out of the 200 cases, 152 were booked cases, which were followed up throughout the pregnancy, 48 cases were un-booked cases.

2. INCIDENCE OF MULTIPLE PREGNANCY IN RELATION TO MATERNAL AGE

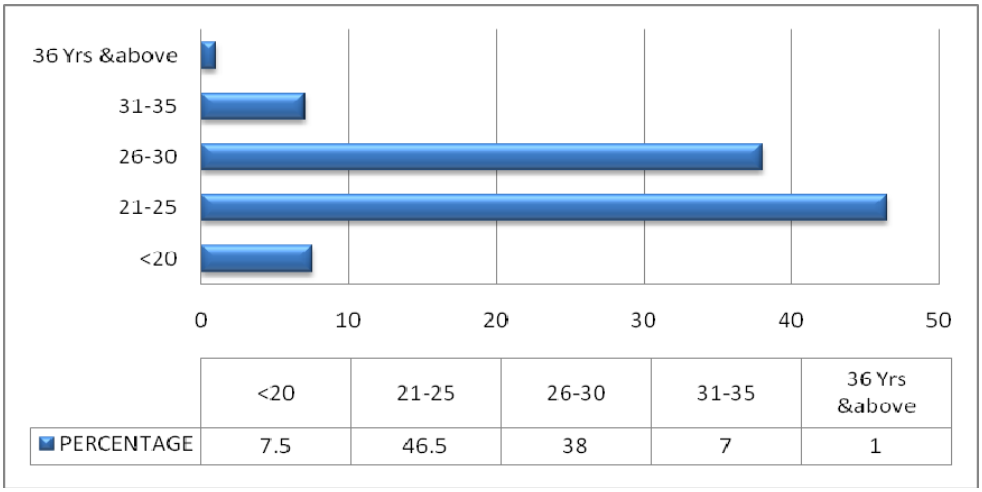
TABLE 3

S.NO	AGE(YRS)	NO.	PERCENTAGE
1.	<20	15	7.5
2.	21-25	93	46.5
3.	26-30	76	38
4.	31-35	14	7
5.	36 Yrs &above	2	1

The most common age group where twins occur, comes in the range of 21-25, forming 46.5%.The next common age group is between 26-30 yrs.

FIGURE - 2

MATERNAL AGE



3. PARITY AND TWIN PREGNANCY

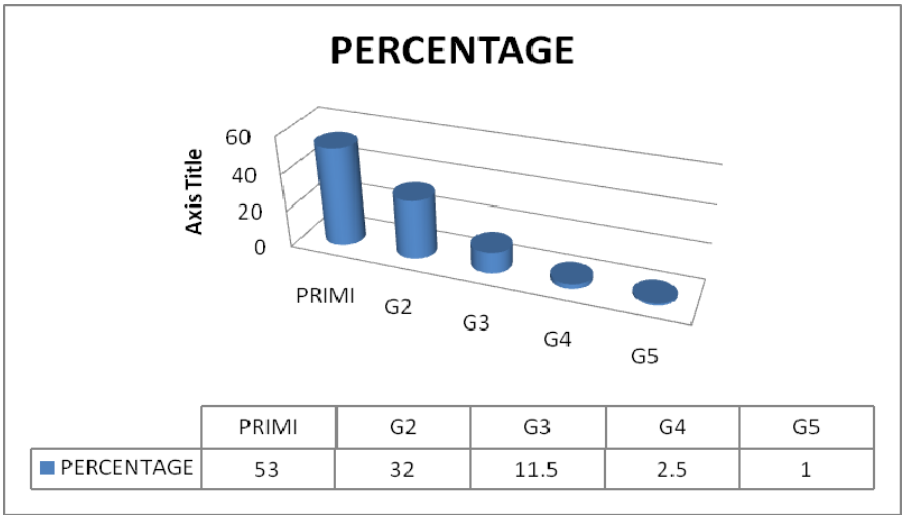
TABLE-4

S.NO	PARITY	NO.	PERCENTAGE
1.	PRIMI	106	53
2.	G2	64	32
3.	G3	23	11.5
4.	G4	5	2.5
5.	G5	2	1

The maximum number of twins occurs in primipara forming a percentage of 53%.The next common is G2 and the incidence decreases as parity increases.

FIGURE-3

INCIDENCE OF TWINS IN RELATION TO PARITY



4. HEREDITARY FACTORS:

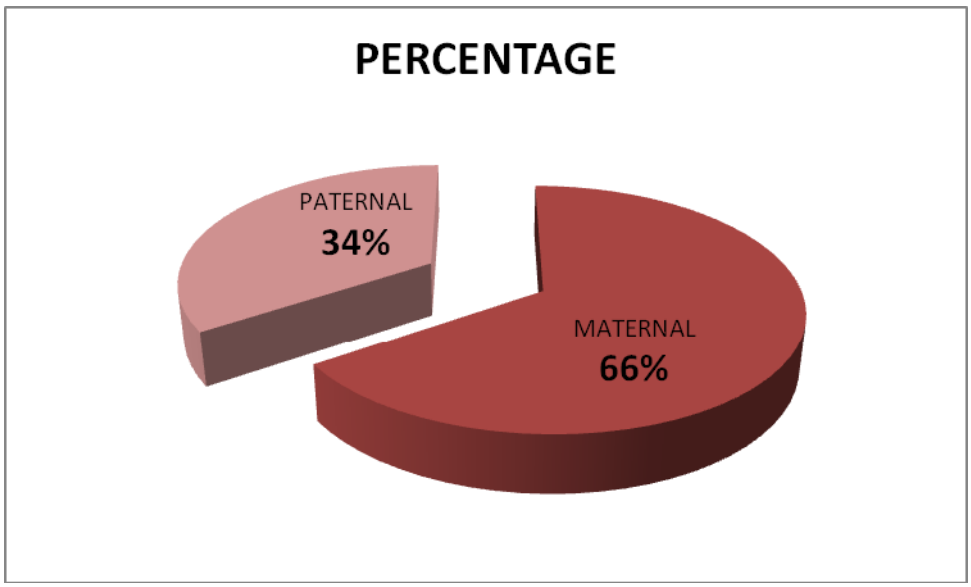
TABLE – 5

S.NO	FAMILY HISTORY	NO.	PERCENTAGE
1.	MATERNAL	40	20
2.	PATERNAL	21	10.5
	TOTAL	**61**	**30.5**

In our study series family history on the maternal side is more associated with twins, than paternal history with 20% on maternal side and 10.5% on paternal side, forming a total of 61 cases with positive family history.

FIGURE-4

INFLUENCE OF HEREDITY



5. INCIDENCE OF SPONTANEOUS Vs INDUCED CONCEPTIONS:

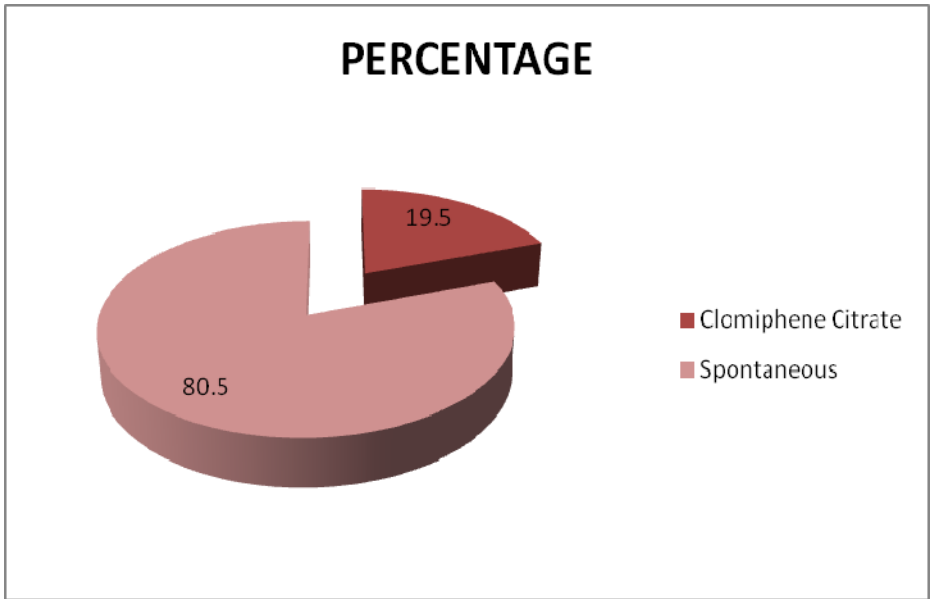
TABLE – 6

S.NO	SPONTANEOUS/ INDUCED CONCEPTION	NO.	PERCENTAGE
1.	Clomiphene Citrate	39	19.5
2.	Spontaneous	161	80.5

The total number of spontaneous conceptions was 161 and 39 were induced pregnancies with clomiphene citrate.

FIGURE-5

INFLUENCE OF OVULATION INDUCTION DRUGS.

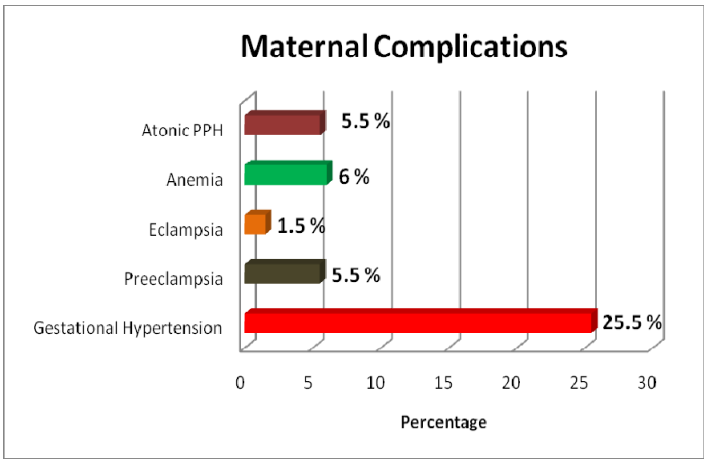


6. MATERNAL COMPLICATIONS:

TABLE-7

S.NO	PREGNANCY COMPLICATIONS	NO.	PERCENTAGE
1.	Gestational Hypertension	51	25.5
2.	Preeclampsia	11	5.5
3.	Eclampsia(imminent and Antepartum)	3	1.5
4.	Anemia(moderate and severe)	12	6
5.	Atonic PPH	11	5.5
	TOTAL	**88**	**44%**

FIGURE-6



Total number of complications was 88. Gestational Hypertension is predominantly higher than the other complications giving a percentage of 25.5%.

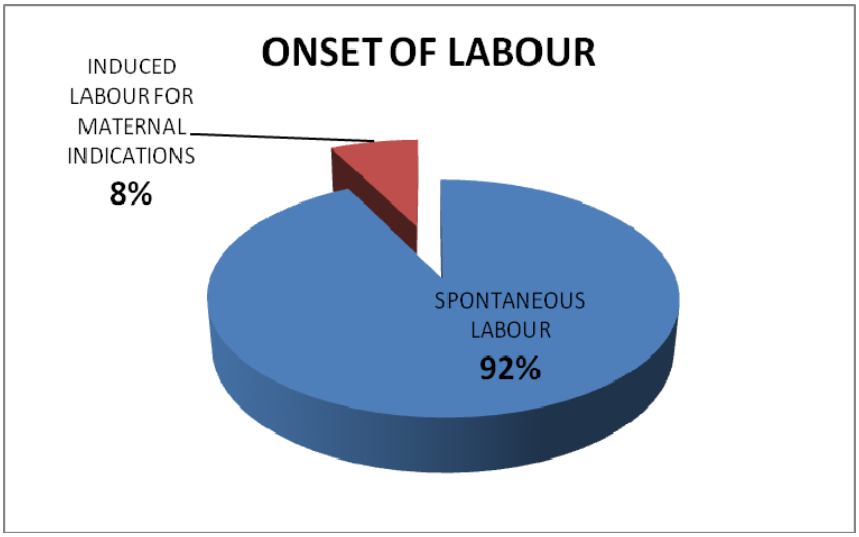
7. SPONTANEOUS/INDUCED LABOUR:

TABLE-8

S.NO	ONSET OF LABOUR	NO.	PERCENTAGE
1.	SPONTANEOUS LABOUR	185	92.5
2.	INDUCED LABOUR FOR MATERNAL INDICATIONS	15	7.5

In our study series, pregnancy was induced in around 7.5% of pregnancies for maternal indications.

FIGURE-7



8. CHORIONICITY

TABLE-9

S.NO	CHORIONICITY	NO.	PERCENTAGE
1.	DCDA	114	57
2.	MCDA	79	39.5
3.	MCMA	7	3.5

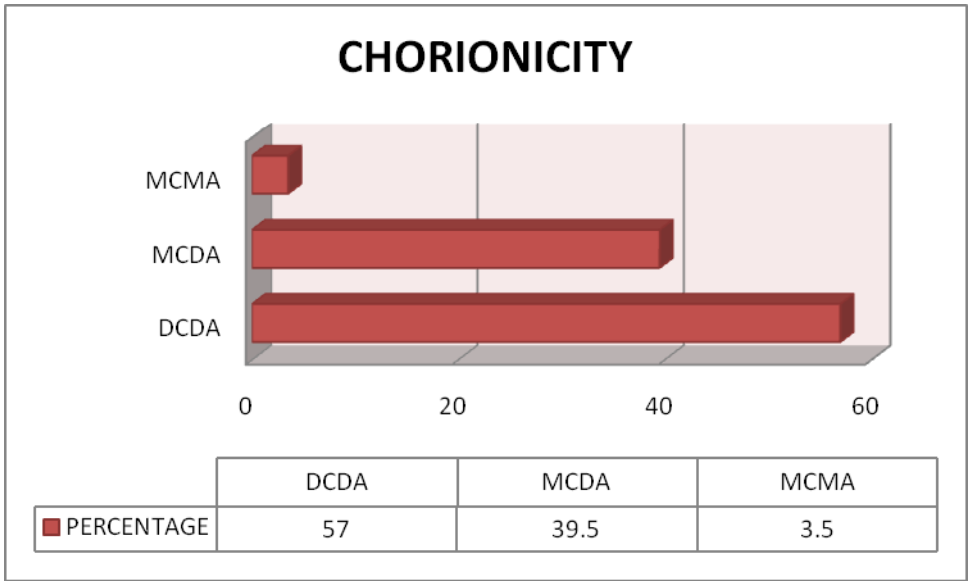
In our study series the percentage of Dichorionic Diamniotic pregnancies were higher of the value 57%.There were 114 - DCDA pregnancies,79 - MCDA pregnancies & 7- MCMA pregnancies.

DCDA – Dichorionic Diamniotic

MCDA – Monochorionic Diamniotic

MCMA – Monochorionic Monoamniotic

FIGURE-8



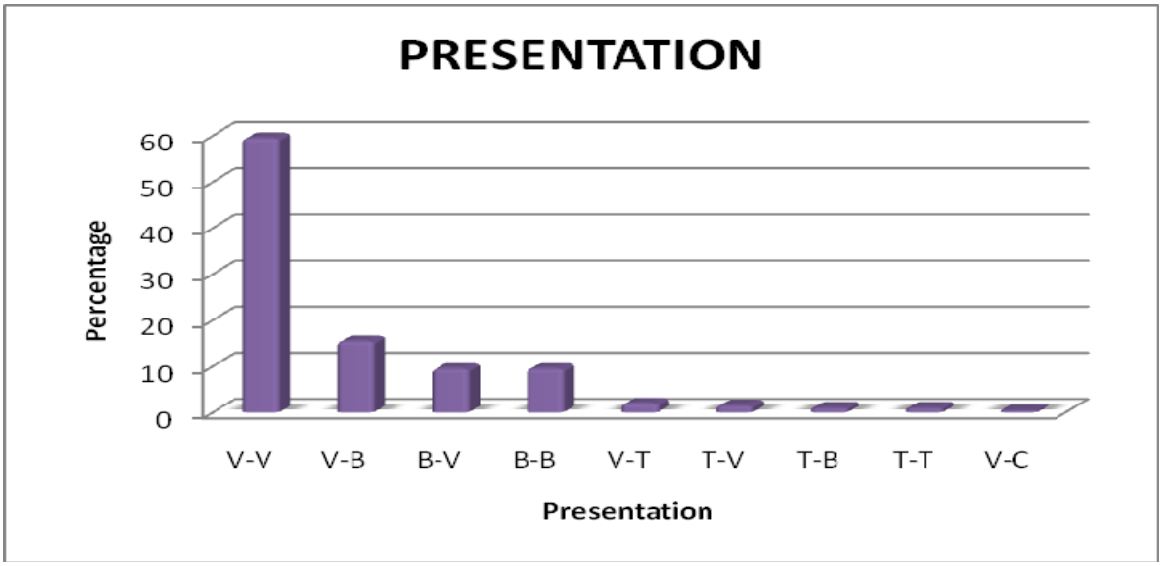
9. PRESENTATION OF BOTH BABIES:

TABLE-10

S.NO	PRESENTATION	NO.	PERCENTAGE
1.	V-V	119	59.5
2.	V-B	31	15.5
3.	B-V	19	9.5
4.	B-B	19	9.5
5.	V-T	4	2
6.	T-V	3	1.5
7.	T-B	2	1
8.	T-T	2	1

9.	V-C	1	0.5

FIGURE-9



The most common presentation in our study is Vertex- Vertex forming a percentage of 59.5%, then comes Vertex- breech presentation-15.5%, least common was vertex-compound presentation- 0.5%.

V – Vertex

B – Breech

T – Transverse

C- Compound presentation

10. GESTATIONAL AGE AT THE ONSET OF LABOUR:

TABLE-11

S.NO	GA IN WEEKS	NO. OF PREGNANCY	PERCENTAGE
1.	28-30	10	5
2.	31-33	22	11
3.	34-36	75	37.5
4.	37 & above	93	46.5

FIGURE-10

In our study majority of the cases delivered preterm < 37 weeks which comes around 53.5% Vs 46.5% above 37 weeks. 5% delivered by 28 to 30 weeks, 11% by 31 to 33 weeks, 37.5% by 34 to 36 weeks**.**

11. PERINATAL MORTALITY AND PERIOD OF GESTATION AT BIRTH:

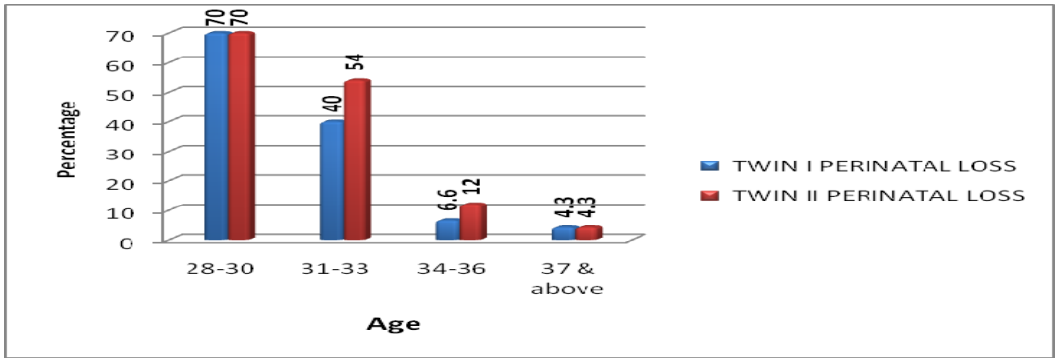
TABLE-12

S.NO	GA IN WEEKS	NO. OF PREGNANCY	TWIN I PERINATAL LOSS	TWIN II PERINATAL LOSS

			NO.	%	NO.	%
1.	28-30	10	7	70%	7	70%
2.	31-33	22	9	40%	12	54%
3.	34-36	75	5	6.6%	9	12%
4.	37 & above	93	4	4.3%	4	4.3%
	TOTAL	**200**	**25**	**60.4%**	**32**	**70.15%**

FIGURE-11

GESTATIONAL AGE AND PERINATAL LOSS



Perinatal loss is inversely related to the period of gestation with much more increased mortality for the 2nd twin. It forms 60.4% in twin I versus 70.15% in twin II.

12. GESTATIONAL AGE OF DELIVERY AND THE PERINATAL OUTCOME WITH RESPECT TO CHORIONICITY:

TABLE-13

GA IN WKS	DCDA NO. & %	LOSS		MCDA NO. & %	LOSS		MCMA NO. & %	LOSS	
		T I	T II		T I	T II		T I	T II
<36	59-51.7%	7	11	42-53.1%	11	12	6-85.7%	3	5
>37	55-48.2%	3	-	37-46.8%	1	4	1-14.2%	-	-
Total	114	10	11	79	12	16	7	3	5

There were 18 cases of loss in DCDA, 23 in MCDA , 8 in MCMA twins < 36weeks & 3 cases of loss in DCDA, 5 in MCDA twins >37weeks.

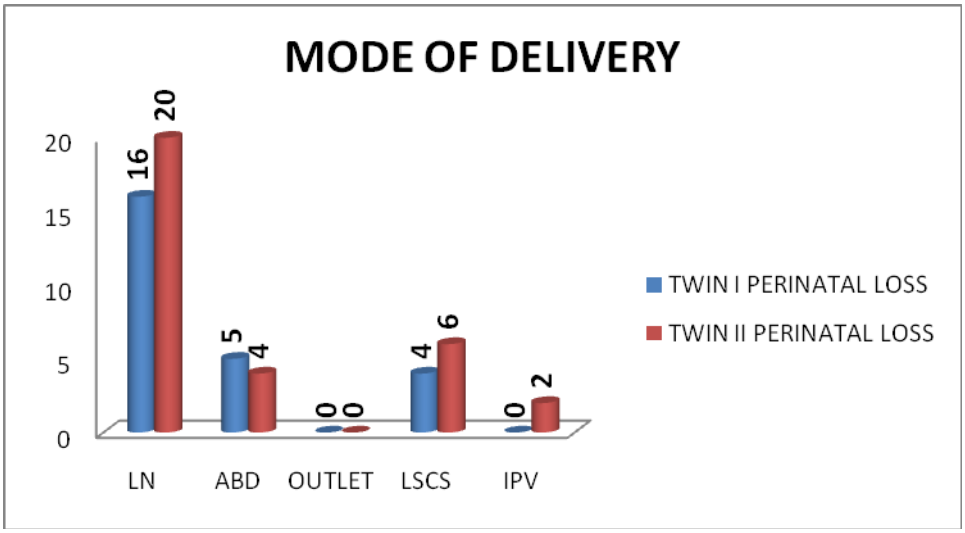
13. MODE OF DELIVERY AND THE PERINATAL OUTCOME:

TABLE-14

S.NO	MODE OF DELIVERY	TWIN I		TWIN II		PERCENTAGE OF PERINATAL LOSS
		NO.	PERINATAL LOSS	NO.	PERINATAL LOSS	
1.	LN	136	16	117	20	14%
2.	ABD	18	5	35	4	16%
3.	OUTLET	2	-	-	-	-
4.	LSCS	44	4	45	6	11.2%

5.	IPV	-	-	3	2	66.6%
	TOTAL	**200**	**25**	**200**	**32**	

FIGURE-12



LN – Labour Natural

ABD – Assisted Breech Delivery

LSCS – Caesarean Section

OUTLET-Forceps delivery

IPV-Internal podalic version

The perinatal loss is maximum for IPV which is around 66.6%, Percentage of loss with ABD-16%, LN-14%, LSCS – 11.2%.

14. DELIVERY TIME INTERVAL BETWEEN THE BABIES AND THE PERINATAL OUTCOME OF THE 2ND TWIN:

TABLE-15

S.NO.	MINUTES	TOTAL	LOSS	%
1.	<10	**153**	**23**	**15%**
2.	11-20	**29**	**5**	**17%**
3.	>21 Minutes	**18**	**4**	**22%**
	Total	**200**	**32**	**54**

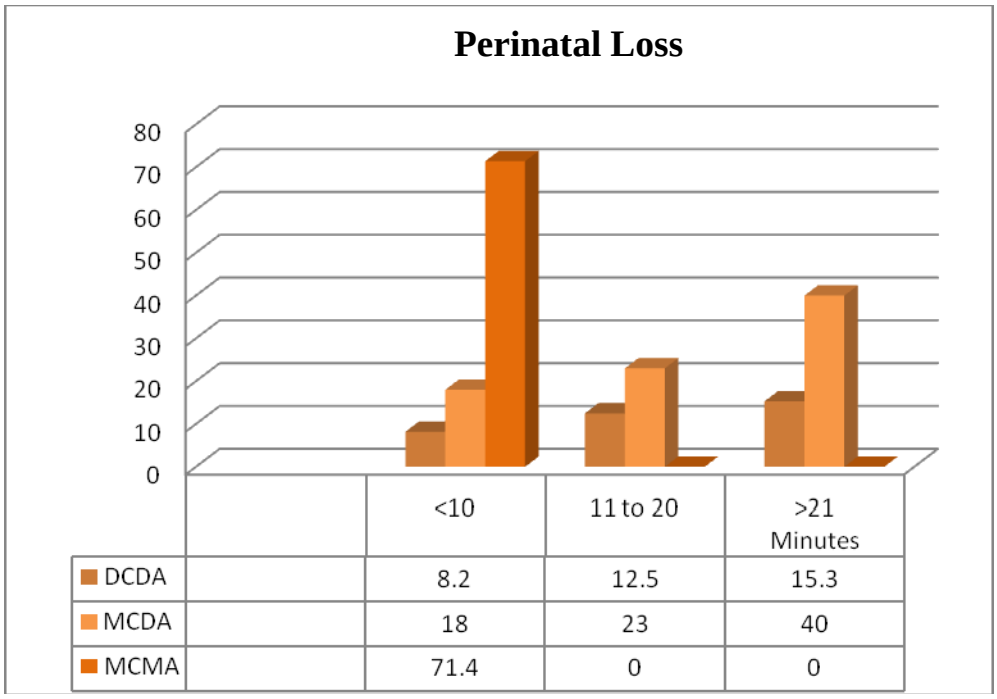
Total number of babies delivered <10 minutes were 153, between 11-20 minutes were 29, >21 minutes were 18. The perinatal loss percentage of babies delivered <10 minutes – 15%, between 11-20 minutes -17%, >20 minutes - 22%.

S.NO	TIME INTERVAL IN MINUTES	DCDA			MCDA			MCMA		
		NO.	LOSS OF TWIN II	%	NO.	LOSS OF TWIN II	%	NO.	LOSS OF TWIN II	%
2.	11-20	16	2	12.5%	13	3	23%	-	-	-
3.	>21 Minutes	13	2	15.3%	5	2	40%	-	-	-

Though majority of the babies deliver within 10minutes, the Perinatal mortality for the 2nd twin is higher as the time interval increases. The loss being more in MCDA pregnancies.

FIGURE-13

PERINATAL LOSS WITH INCREASING INTER TWIN DELIVERY INTERVAL



15. BIRTH WEIGHT AND THE PERINATAL OUTCOME:

TABLE-16

S.NO	BIRTH WEIGHT IN KG	TWIN I		TWIN II		PERCENTAGE OF PERINATAL LOSS
		NO.	PERINATAL LOSS	NO.	PERINATAL LOSS	
1.	<1 kg	9	8	13	13	94%
2.	1-1.5	25	10	39	13	46%
3.	1.6-2	82	3	82	4	4.2%
4.	2.1-2.5	55	4	50	1	4.5%
5.	>2.5	29	0	16	1	3.1%

	TOTAL	**200**	**25**	**200**	**32**	**75.9%**

FIGURE-14

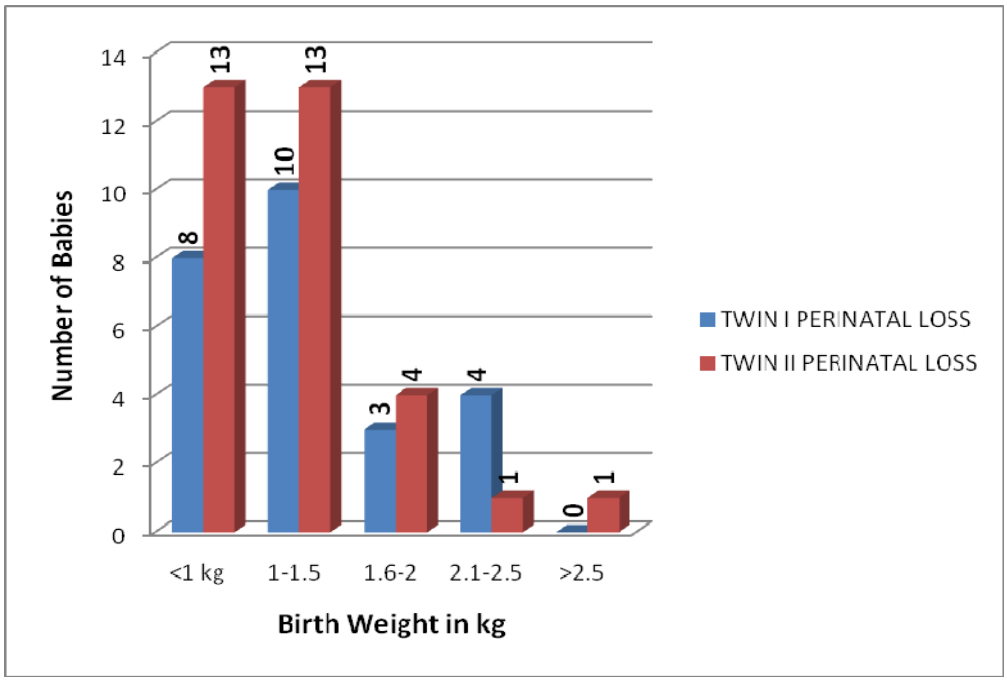
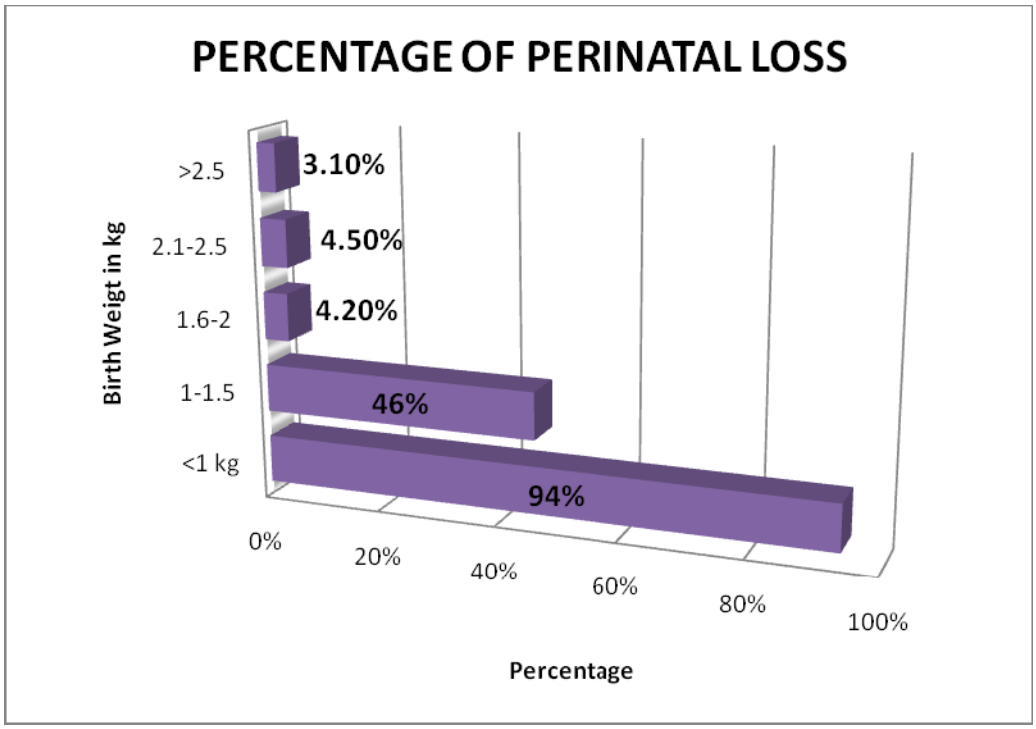


FIGURE-15



Perinatal mortality is highest in the birth weight of <1.5 kg. The loss percentage for babies <1 kg - **94%** , 1 - 1.5 kg – **46 %** , 1.6 – 2 kg **-4.2 %** , 2.1 - 2.5 kgs **-4.5 %** , > 2.5 kgs **-3.1 %** .

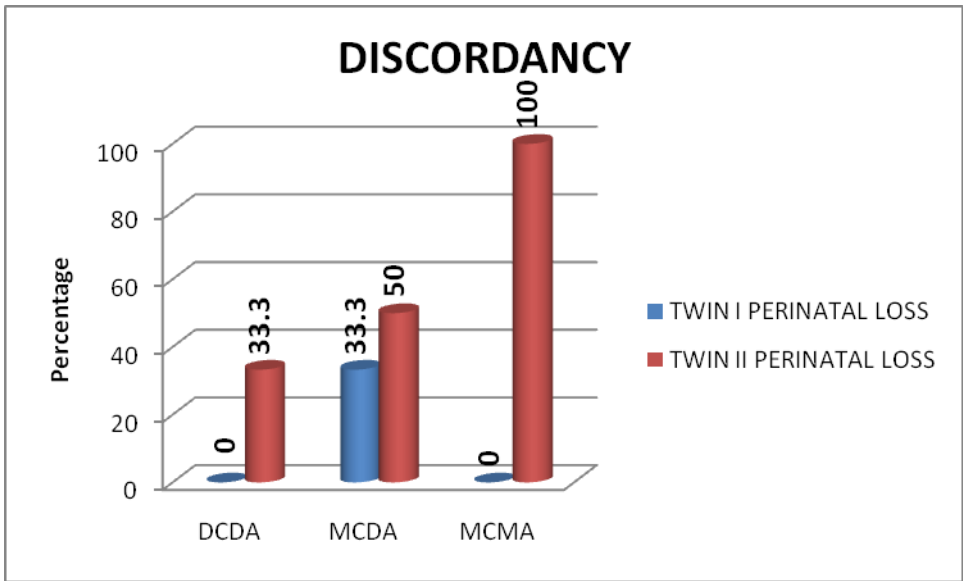
16. DISCORDANCY AND ITS ASSOCIATION:

TABLE-17

S.NO	CHORIONICITY	TWIN I			TWIN II		
		TOTAL NO.	LOSS NO.	LOSS%	TOTAL NO.	LOSS NO.	LOSS%
1.	DCDA	3	-	-	12	4	33.3%
2.	MCDA	6	2	33.3%	12	6	50%
3.	MCMA	-	-	-	2	2	100%
	TOTAL	9	2	22.2%	26	**12**	**46.15%**

From our study series, Discordancy is high in MC twins (20) than DC twins(15), loss percentage is 22.2% in 1st twin and 46.17% in 2nd twin and the mortality for the discordant 2nd twin in a MC pregnancy is still higher.

FIGURE-16



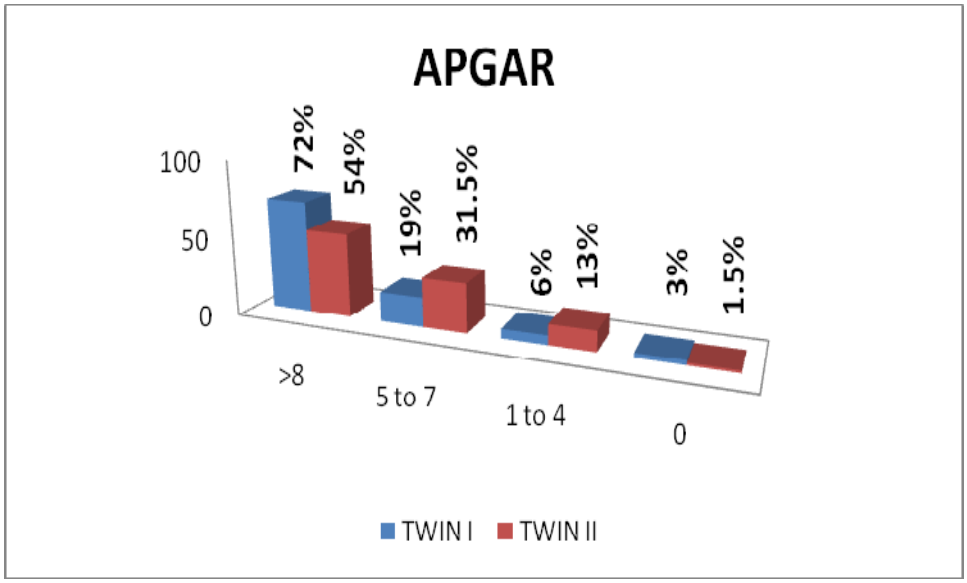
17. APGAR SCORES:

TABLE-18

S.NO	APGAR	TWIN I		TWIN II	
		NO.	PERCENTAGE	NO.	PERCENTAGE
1.	>8	144	72	108	54
2.	5 – 7	38	19	63	31.5
3.	1 – 4	12	6	26	13
4.	0	6	3	3	1.5

The APGAR scores for the 2nd twin are lower than the 1st twin.

FIGURE-17



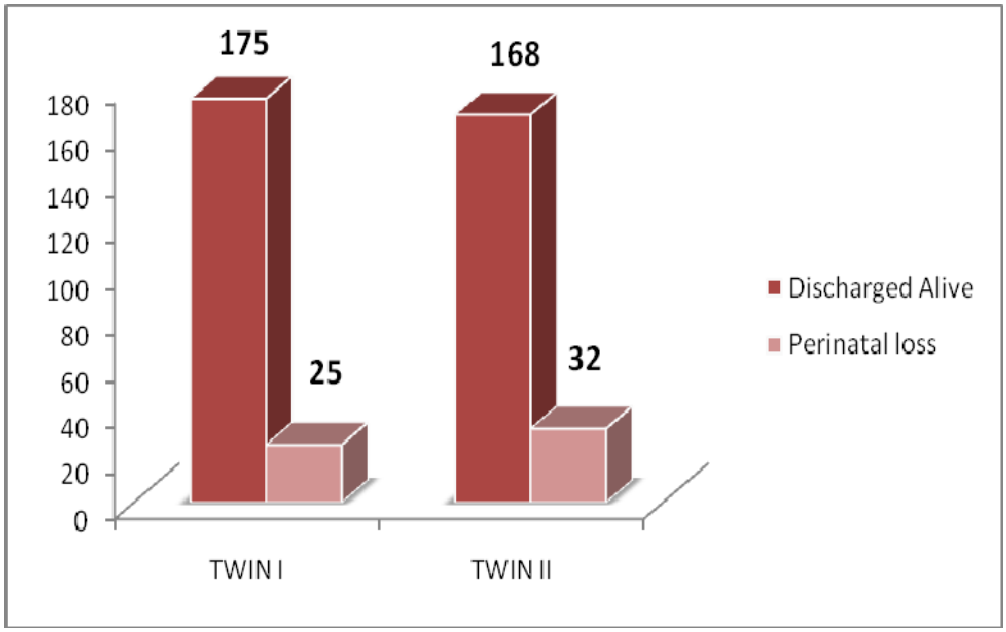
18. PERINATAL MORTALITY RATE:

TABLE-19

S.NO	OUTCOME	TWIN I	TWIN II
1.	Discharged Alive	175	168
2.	Perinatal loss	25	32
3.	Perinatal mortality rate	125/1000	160/1000

Mortality for the 2nd twin- 160/1000 is higher than the 1st twin- 125/1000. Of the total number of cases, 175 babies were discharged alive in twin I Vs 168 babies of twin II. Out of total perinatal loss of 57 babies, 32 were the 2nd of the twin.

FIGURE-18



19. CAUSES OF DEATH IN BOTH TWINS:

TABLE-20

S.NO	CAUSE OF DEATH	TWIN I	TWIN II
1.	BIRTH ASPHYXIA	5	10
2.	SEPSIS	5	8
3.	CORD PROLAPSE	2	1
4.	PREMATURITY AND ITS COMPLICATIONS	3	4
5.	ANAMOLOUS BABY	3	-
6.	IUGR	3	5
7.	NNS	1	3
8.	IUD	2	-
9.	TTTS	1	1
	TOTAL	25	32

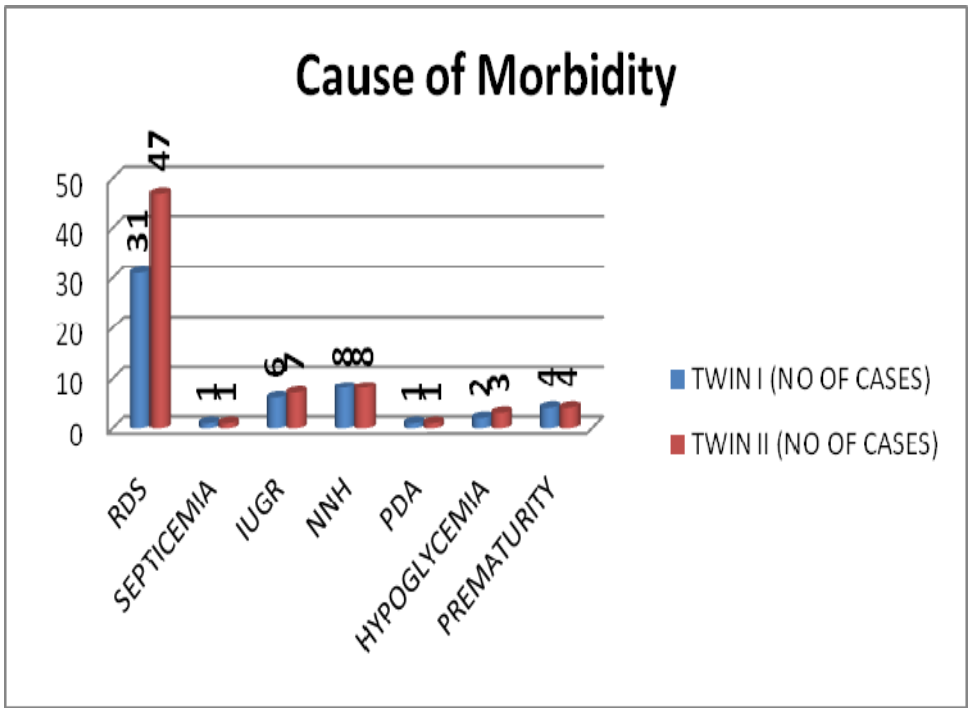
NNS-Neonatal seizures, IUD-Intrauterine death, TTTS-Twin–to Twin Transfusion syndrome

20. CAUSES OF MORBIDITY IN BOTH TWINS:

TABLE-21

S.NO	CAUSES OF MORBIDITY	TWIN I (NO. OF CASES)	TWIN II (NO. OF CASES)
1.	RDS	31	47
2.	SEPTICEMIA	1	1
3.	IUGR	6	7
4.	NNH	8	8
5.	PDA	1	1
6.	HYPOGLYCEMIA	2	3
7.	PREMATURITY AND ITS COMPLICATIONS	4	4
	TOTAL	**53**	**71**

FIGURE-19



RDS – Respiratory Distress Syndrome

PDA-Patent ductus arteriosus

IUGR – Intra Uterine Growth Restriction

NNH – Neonatal Hyperbilirubinemia

RDS, IUGR &Hypoglycemia are higher in 2nd twin.

21. FINAL COMPARISON OF OUTCOME OF THE BABIES:

TABLE-22

S.NO	OUTCOME	DCDA		MCDA		MCMA	
		I	II	I	II	I	II
1.	NO. OF BABIES	114	114	79	79	7	7
2.	MEDIAN GA	36 WEEKS		35 WEEKS		33 WEEKS	
3.	AVERAGE BABY WEIGHT	2	1.9	1.9	1.7	1.4	1.3
4.	5 MINUTE APGAR < 7	18	41	34	44	4	7
5.	AVERAGE LENGTH OF NICU STAY	2.06	3.8	2.5	4.1	1.5	4.1
6.	NO. OF NICU ADMISSIONS	35	52	31	41	6	7
7.	DISCHARGED ALIVE	104	103	67	63	4	2
8.	EXPIRED (INCLUDES STILL BIRTHS)	10	11	12	16	3	5
9.	PNMR/1000 BIRTHS	87.7	96.4	151.8	202.5	428.5	714.2

The median GA in DCDA twins were 36 weeks, MCDA – 35weeks, MCMA – 33 weeks. Average Birth weight is approximately 100 grams higher in DC twins than in MC twins. The average length of NICU stay and the no. of

NICU admissions were higher in MC twins than in DC twins raising the mortality of MC twins to 177/1000 births Vs 92/1000 births in DC twins.

FIGURE-20

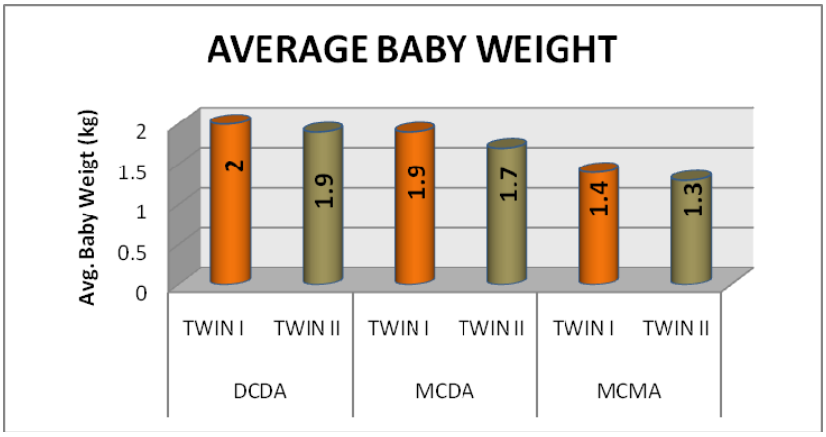


FIGURE-21

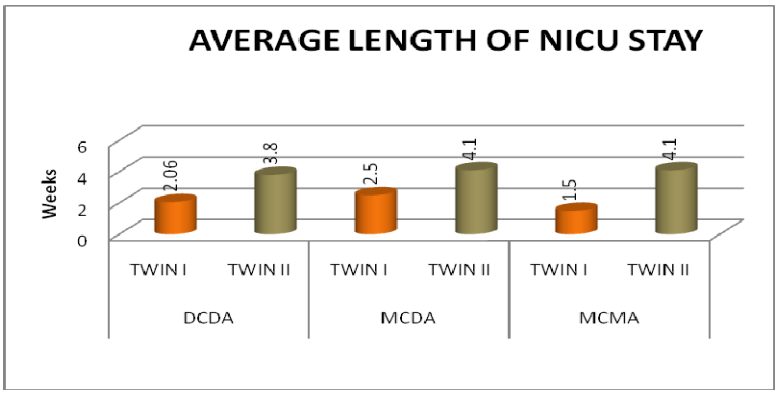


FIGURE-22

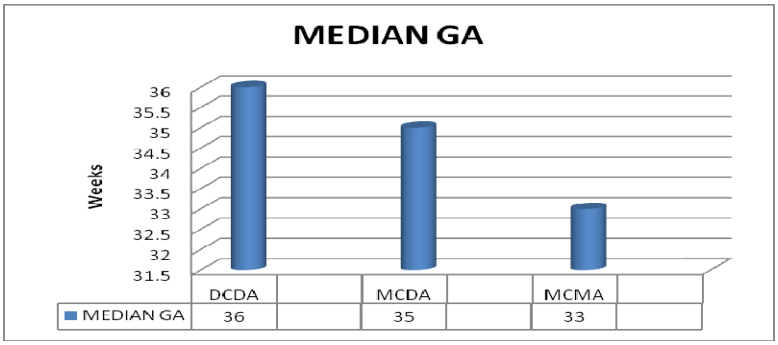


TABLE- 23

S.NO	OUTCOME	TWIN I	TWIN II
1.	5MINUTE APGAR <7	56	92
2.	NO. OF NICU ADMISSIONS	72	100
3.	DISCHARGED ALIVE	175	168
4.	EXPIRED	25	32
5.	PNMR	125/1000	160/1000

5 minute APGAR < 7 was observed in 56 of twin I Vs 92 of twin II, number of NICU admissions were high for twin I -100 Vs 72 of twin II.

22. MORBIDITY IN COMPARISON WITH MC AND DC TWINS:

TABLE-24

S.NO	MORBIDITY	DCDA		MCDA		MCMA	
		I	II	I	II	I	II
1.	RDS	17	29	12	17	2	1
2.	SEPTICEMIA	1	1	0	0	0	0
3.	IUGR	0	0	5	6	1	1
4.	NNH	6	6	2	2	0	0
5.	PDA	1	1	0	0	0	0
6.	HYPO GLYCEMIA	1	3	1	0	0	0
7.	PREMATURITY	1	1	3	3	0	0
	TOTAL	27	41	23	28	3	2

Morbidity due to prematurity and IUGR is higher in MC twins.

MORBIDITY DUE TO VARIOUS CAUSES BETWEEN TWIN I & II

TABLE-25

S.NO	CHORIONICITY	TWIN I	TWIN II
1.	DCDA	27	**41**
2.	MCDA	23	**28**
3.	MCMA	3	**2**
	TOTAL	53	**71**

23. MORTALITY IN COMPARISON WITH MC AND DC TWINS:

TABLE-26

S.NO	CAUSE OF DEATH	DCDA		MCDA		MCMA	
		I	II	I	II	I	II
1.	SEPSIS	2	3	3	4	0	1
2.	IUGR	0	0	**2**	**4**	1	1
3.	BIRTH ASPHYXIA	4	3	1	5	0	2
4.	ANOMOLOUS BABY	1	0	1	0	1	0
5.	CORD PROLAPSE	1	1	1	0	0	0
6.	PREMATURITY	0	1	**2**	**2**	1	1
7.	TTTS	0	0	**1**	**1**	0	0
8.	NNS	1	3	0	0	0	0
9.	IUD	1	0	1	0	0	0
	TOTAL	10	**11**	12	**16**	3	**5**

In the study series IUGR, and prematurity as a cause of death is more predominant with MC twins than DC twins. TTTS has resulted in the death of 2 babies of MC twins.

TOTAL DEATHS IN TWIN I & II:

TABLE-27

S.NO	CHORIONICITY	TWIN I	TWIN II
1.	DCDA	10	**11**
2.	MCDA	12	**16**
3.	MCMA	3	**5**
	TOTAL	25	**32**

DISCUSSION

INCIDENCE:

The incidences of twins have increased worldwide. This is due to the widespread use of ART methods and the advanced age of motherhood.

The incidence in our study series has increased when compared with June 2010-July 2011 with June 2011-July2012 from 13.4 to 16.3/1000 births. This increase in incidence is probably due to the generous use of ovulation induction drugs.

A similar hike was seen in a study by Karien Hack[14] in the year 2008,where he proposed that in Netherlands, the number of twins born in the early 20th century was 12.4/1000 births. This had increased to 17.8 twins per 1000 births by the end of the 20th century.

MATERNAL AGE AND PARITY:

In our study series, the common age group was 21-25 yrs forming a percentage of 46.5% followed by 38% in the age group of 26-30 years. This is in comparison with a study by A.G.W. Farrell et.al(1963)[15] where they found a maximum incidence of 30.2% in the age group of 26-30 years and 26.7% between the age group of 21-25 years.

In our study, twin incidence was found to be higher in primipara of 53%, G2- 32%, G3- 11.5%, G4- 2.5%, G5- 1%. With increasing parity the incidence of twinning has reduced in our study.

This is in association with Ji Young Kwon et.al (2011)[16] where they found the incidence of 67.1% in the nulliparous women. In contrast with our study, Anna Dera et.al[17] from Poznan Medical University between the years 2004-2007 quoted the incidence to be maximum of 40.6% in G5, followed by 25% in G2, 14% in G4, 12.5% in primi & 7.8% in G3.

FAMILY HISTORY:

In our study series, we find that a family history of twinning on the maternal side in 20% of cases in comparison with 10.5% on the paternal side, hence a positive family history on the maternal side has more significance. This has been in correlation with White & Wyshak in 1964[18] where they quote that, Woman who are actually dizygotic twins themselves, gave birth to twins at an increased frequency than a positive history on the paternal side, the incidence of the former being 1 in 58 births, versus 1 in 116 pregnancies of the latter.

MATERNAL COMPLICATIONS:

In our study series, out of the various complications, Gestational Hypertension was predominantly higher than the other complications giving a percentage of 25.5%, followed by anaemia-6%,preeclampsia & atonic PPH-5.5,eclampsia – 1.5%. This is on the higher side compared with singleton pregnancies. The incidence of preeclampsia is 2.6 times higher in twins than in singleton pregnancies. (ACOG practice bulletin 2004)[19]

In comparison with another study by Apichart Chittacharoen et el (2000)[20], they found an incidence of anemia in 21.5%, PIH – 13.4%, PPH – 15.6%. In another study by Naushaba Rizwan et al (2009)[21] incidence of PIH was similar to our study – 31.2%, with an increased incidence of anemia – 65.6%.

PRESENTATION OF THE FETUSES:

The most common presentation in our study is Vertex- Vertex forming a percentage of 59.5%, then comes Vertex- breech presentation. This is in association with study by Apichart Chittacharoen et.al (2000)[20], Chervenak FA et.al (1984)[22] and A.G.W. Farrell et.al (1963)[15]

GESTATIONAL AGE AT THE ONSET OF LABOR:

In our study majority of the cases delivered preterm < 37 weeks which comes around 53.5% Vs 46.5% above 37 weeks. The most important cause of perinatal morbidity and mortality in a twin pregnancy is due to the prematurity and its complications[20]. Apichart Chittacharoen et.al from Ramathibodi Hospital in the year 2000, observed per-term delivery in 49.2% cases. Similar observations have been cited by kauppila A et al (1973)[23], Newton ER et al (1986) [24], Gardner MO et al (1986) [25], Buscher U et al (2000) [28].

In a study by Anna dera et al[17] (2007) they observed preterm delivery of <37 weeks in 62.5% vs 37.5% of deliveries >37weeks.

In relation to chorionicity, in our study the median GA in DCDA, MCDA & MCMA twins were, 36, 35 & 33 weeks respectively implies preterm delivery to be more common with MC twins than DC twins. The median GA in DC twins is 1 week longer than that in MC twins.

Continuing pregnancy >37weeks in a MC pregnancy has a higher mortality when compared with DC pregnancies, the perinatal loss in our study in DC twins >37weeks- 3 Vs 5 babies in MCDA delivering >37 weeks.

A similar association has been quoted in a study by Hack and co workers[14] (1907-1938), where it is said that DC twins continue with intrauterine life 1 week (36 weeks) longer than MC twins (35 weeks).In study by Apichart Chittacharoen et al[20], where they cited that preterm delivery <32 weeks was higher in MC twins (9.2%) than in DC twins (5.5%).

Similar to our study, another study published in BJOG 2011[29] said that in MCDA pregnancies after 32 weeks of gestation, there were 6 neonatal deaths. Perinatal mortality was 7/1000 births in those who delivered >37 weeks .Hence they conclude that mortality at term was higher in MC twins than in DC twins hence waiting for spontaneous onset of labor after 37 weeks is not justified. Planned elective delivery between 36-37 weeks should be considered, which avoids the respiratory disorders in the neonate due to preterm delivery. Also the 1% risk of IUD after 37weeks can be avoided. This does not warranty elective caesarean section in all cases & does not have significant impact on the neonatal outcome.

Results from another study by Smith NA (2008), Wilkins-Haug Letc in Brigham women's hospital, Boston[31] suggests that most of the MCDA pregnancies complicated by discordancy & TTTS ends up in preterm births and IUFD. In the absence of such complications, elective preterm delivery is not indicated.

An article published in BJOG 2012[32] states that in The Australian study, in uncomplicated twin pregnancies delivered electively by 37 weeks had lesser incidence of adverse outcomes in the neonate compared with those pregnancies with >37 weeks with awaited spontaneous onset of labour.

MODE OF DELIVERY AND PERINATAL OUTCOME:

In our study series the perinatal loss is maximum for Internal podalic version of the 2nd twin and breech extraction, done in 3 cases which is around 66.6%

On further research regarding with entire event, the 1st case was DCDA twins in 37 weeks, 1st twin delivered by LN with a birth weight of 1.16kgs, 2nd twin delivered by IPV and breech extraction 60mints from the 1st twin's birth and had a birth weight of 2.3 kgs. Both babies had good APGAR and hence were discharged in good health. Whereas in the other 2 cases of stillborn second twins, they were MCDA twins of GA< 32 weeks, the delivery time interval was 180 minutes which is thrice the time of the pervious case, both the 1st twin's weight was <1.2 kgs and 2nd twin's weight was 1 kg. 1st of the twin's in both cases died due to LBW, sepsis and prematurity.

Though the "P" value is 0.014 which is statistically significant, on taking the associated factors in those 2 cases of still born babies after IPV, the factors like chorionicity, delayed time interval between the babies, prematurity, LBW etc, are more responsible for the death of the babies rather than the procedure per se. A.G.W.Farrell et al at Edendale hospital[15] says that internal podalic version has the highest mortality to the 2nd twin and it should be reserved as the last option when external podalic version fails.

But the loss with each mode of delivery in the 2nd twin was LN - 17%, ABD - 11.4%, LSCS - 13%, was not statistically significant. Hence the perinatal outcome of the 2nd of the twin was not affected by the mode of delivery.

Various other studies gave varied inferences with majority of them stating that perinatal outcome of the 2nd twin was not being affected by the mode of delivery.

In a Bombay Hospital Journal, Vol. 51(2009)[33], they found the loss of 2nd twin to be higher with breech delivery - 33.3%, vaginal delivery - 29.72%, instrumental delivery - 28.57%, LSCS - 10.63%

In a study by Bjelic-Radisic V et al in 2007[34],he stated that low APGAR was found maximum in those cases delivered by V-CS (vaginal-1st twin, CS of 2nd twin), followed by V-Vaginal breech, then CS-CS, he says that mortality is

higher in non-vertex 2nd twin. The high CS rate in V/NV presentation and the significantly worse perinatal short-term outcome of NV second twins after VB of the first twin, underlines that randomized studies are necessary to evaluate the best delivery mode for V/NV twins.

He reported an incidence of 5% of 2nd twin delivered by CS. In our study we had only one case of 2nd twin being delivered by CS after vaginal birth of 1st twin, and the mortality was 100%. To elaborate, that was a DCDA pregnancy in 36weeks of GA, after vaginal delivery of 1st twin emergency LSCS was done for compound presentation of 2nd twin, the time delay was 60 minutes, the birth weight of 1st twin was 2.6 kgs and it had a good APGAR, the 2nd twin had APGAR <5 and weighed 2.9 kgs and the baby expired.

A study in the Poznan Medical University between 2004- 2007[17] suggested that, mode of delivery has no influence on the morbidity & mortality of the non cephalic 2nd twin of weight >1.5 kgs.

This was supported by Usta I.M in 2004[36] suggested twins >1.5kgs to be delivered vaginally and the others by LSCS. Fishman in 1993[35] stated that no excessive morbidity or mortality was associated with vaginal breech delivery of 2nd twin. Another article by Charlotte N et.al[38] suggests that there is no consensus regarding the ideal route of delivery for non-vertex twins. They state

that, it is ideal to do LSCS for non vertex 1st twin since the phenomena of interlocking twins is seen with breech/ vertex twins.

On the contrast, Yang in 2005[37] stated that vaginal delivery causes more morbidity to the 2nd neonate than caesarean section of both the twins.

ACOG practice bulletin (2004)[19] does not give a clear cut conclusion regarding the mode of delivery.

Cochrane review published in 2011[39] in regarding this issue states that, delivering non vertex 2nd twins by vaginal route has association with increased maternal morbidity but does not improve the neonatal outcome, hence further trials are needed to conclude regarding opting for LSCS.

In a BJOG journal regarding MCDA pregnancies published in 2011[29], they state that perinatal mortality were similar between all modes of delivery groups.

TIME INTERVAL BETWEEN DELIVERIES OF THE BABIES:

In our study series, total number of babies delivered <10 minutes were 153, between 11-20 minutes were 29, >21 minutes were 18. The perinatal loss percentage of babies delivered <10minutes – 15%, between 11-20 mins-17%, >20 minutes - 22%.

Similar observations were quoted in a Bombay Hospital Journal, Vol. 51[33], perinatal loss for 2nd twin was, 10mins- 10.9%, 11 – 20 minutes – 16.6%, >21 minutes – 66.6% .

In our study series though majority of the babies deliver within 10minutes, the Perinatal mortality for the 2nd twin is higher as the time interval increases. The loss being more in MCDA pregnancies 40% Vs 15.3% in DC pregnancies delivered >21 minutes. The "P" value is 0.000, hence it is statistically significant.

Ji Young Kwon, Won Sik Yoon, from The Catholic University of Korea, in 2011[16] observed a better neonatal outcome when the inter twin delivery time interval was < 10 minutes.

BIRTH WEIGHT AND DISCORDANCY:

It is a known fact that birth weight less than 1500 gms has a poorer outcome in terms of morbidity and mortality of both the twins. In our study series, the Perinatal mortality is highest in the birth weight of <1.5 kg, giving a "P" value of 0.000, hence it is statistically significant. The loss percentage for babies <1 kg - **94%** , 1 - 1.5 kg – **46 %** , 1.6 – 2 kg **-4.2 %**, 2.1 - 2.5 kgs **-4.5 %** , > 2.5 kgs –**3.1 %.**

Outcome	DCDA		MCDA		MCMA	
	I	II	I	II	I	II
Average Birth weight in kgs	2	1.9	1.9	1.7	1.4	1.3

The average Birth weight is approximately 100 grams higher in DC twins than in MC twins.

Similar observation cited by Hack and Co-workers[14] where he compared MC and DC twins in 651 pairs between 1907 – 1938, and he observed that birth weight of DC twins were 288 grams higher than MC twins.

From our study series, Discordancy is high in MC twins than DC twins; loss percentage is 22.2% in 1st twin and 46.17% in 2nd twin. And the mortality for the discordant 2nd twin in a MC pregnancy is still higher,
(50% vs. 33.3% in DC twins). The "P" value is 0.164 hence the difference is not statistically significant.

Percentages of discordant babies were equal in both MC & DC twins. But the mortality was higher for monochorionic pregnancies (33.3%) than dichorionic (15.7%) pregnancies (Hack and Co-workers) [14]

Similar to our study was Pawel Krajewski et al in 2008[40], stated the mean birth weight of 2nd twin was lower than the 1st twin, 2.17kgs Vs 2.29kgs with a p value of 0.049.

APGAR SCORES AND ITS INTERPRETATION:

In our study series the outcome of the 2nd twin was judged based on differences in APGAR score of both twins. There were 28% of 1st twin Vs 46% of 2nd twin with APGAR <7. Numbers of NICU admissions were high for twin II -100 Vs 72 of twin I. The "P" value is 0.094 hence this difference is not statistically significant.

The perinatal mortality is around 142/1000 live births in twins which is almost twice that in a single tone pregnancy which comes around 74/1000 live births. Mortality for the 2nd twin - 160/1000 is higher than the 1st twin-125/1000. Of the total number of cases, 175 babies were discharged alive in twin I Vs 168 babies of twin II. Out of total perinatal loss of 57 babies, 32 were the 2nd of the twin.

Similar observation was seen in a comparative analysis of both twins by Pawel Krajewski et.al[40] from neonatology dept, Poland. Mean APGAR were

significantly (p=0.017) lower in 2nd of the twins compared with the 1st twin. PNMR in their study in 2nd twin – 7.9% Vs 1st twin – 2.3%.

CAUSE OF DEATH IN THIS STUDY:

Most common cause of death in this study is Birth Asphyxia due to prematurity, IUGR. To classify them we observed 15 deaths due to birth asphyxia,13 cases due to sepsis, 3 cases of cord prolapse, 7 cases due to prematurity, 3 anomalous babies, 8 babies due to IUGR, 4 babies had neonatal seizures, intra uterine demise in 2 cases & TTTS in 2 (MC twins) cases. In our study series, we observed IUGR (6 Vs 0), prematurity (4 Vs 1) and sepsis (7 Vs 5) being higher in MC twins than in DC twins.

In Apichart Chittacharoen el study[20], most common neonatal complication LBW- 62.3%, prematurity was the most common cause of death, they observed no neonatal death in those cases delivered after 34 weeks.

Pawel Krajewski et al[40] cited that the most common morbidity in 1st twin was RDS, NNH and sepsis. Hypoglycemia being more common in 2nd twin, this was similar to our study were we observed 3 cases of 2nd twin to be hypoglycemic Vs 2 cases of 1st twin.

In our study we had 2 babies of 1st twin IUD, in both cases the pregnancy was complicated by PIH. One case was DCDA, induced by 35 weeks for severe PIH, the discordant 2nd twin delivered vaginally with birth weight of 1.6 kgs (1st twin dead born, b.wt - 2.5kgs). The 2nd twin was in NICU for RDS and was discharged in good health.

In the second case it was MCDA pregnancy complicated by TTTS, pregnancy was terminated by 32 weeks, with discordant 2nd twin weighed 1.16kgs. The APGAR score was <5 and hence the baby expired.

In comparison with other studies, HHN Woo et.al [41] in 2000, observed 4 cases of 1st twin IUD out of 182 twin pregnancies, he observed 83% of cases were MC twins with evidence of TTTS. All the babies delivered by 34 weeks. The 2nd twin all 3 cases had good APGAR and had an uneventful neonatal period, except in one case which was complicated by IUD of the 2nd twin by 30 weeks (1st twin died in utero by 19 weeks). All cases were complicated by PIH in the mother, they concluded that single fetal death by 2nd and 3 rd trimester is associated with high mortality for the surviving twin especially in MC twins.

SUMMARY

The perinatal mortality and morbidity is higher for twins when compared with singletons. Monochorionic twin pregnancies are at a higher risk than dichorionic twin pregnancies and the perinatal complications are more.

The total deliveries at Raja Mirasudhar hospital ,Thanjavur Medical college during the study period June 2010 to July 2012 was 14300.Total twin deliveries during this period was 234 cases. Out of the 234 cases only 200 cases were taken for the study.

Incidences of twin pregnancy with various maternal factors were studied, of which twin pregnancies were common in the age group of 21 – 25 yrs, and common in Primi.

The incidence of maternal complications were varied in a multiple pregnancy. 44% of women had complications due to twin gestation, off- which gestational hypertension was highest of around 25.5%. With respect to chorionicity, there were 114 DCDA twins, 79 MCDA, twins, 7 MCMA twins.

On comparing the perinatal mortality between both twins, it was found that the 2nd twin is at a higher risk than the 1st one.

The PNMR for 2nd twin versus 1st twin was, 125/1000 live births Vs 160/1000 live births. Birth weight and GA at delivery are the two factors which decide the perinatal outcome than the chorionicity.

LBW babies were 171 out of 200; VLBW babies were 34 out of 200 cases. The perinatal loss was maximum in B.wt < 1kg forming 96%.

The number of babies lost weighing <2.5 kgs were 56, out of which 31 were 2nd of the twins.

The respective Median GA of delivery was 36, 35, 33 weeks for DCDA, MCDA, MCMA pregnancies in this study.

Incidence of preterm deliveries < 37 weeks were 53.5% and the perinatal loss was 12.25% (total of 49 babies) in both twins. Out of the 49 babies, 28 were the 2nd twin, implying that the 2nd twin had a bad prognosis.

The most common presentation of the twins was vertex- vertex in 59.5% of cases. The most common Mode of Delivery was Labour Naturale of both the twins. Mortality was higher for IPV & Assisted breech delivery. Out of 3 IPV, 2

babies expired. The loss was also due to VLBW of the babies and the prolonged delivery time interval between the babies.

Longer Delivery time interval between the babies increases the perinatal mortality of the 2nd twin, mortality in those delivered <10 minutes, 11-20 minutes, >21 minutes were 15.03%, 17.2%, 26.6% respectively. Hence the ideal time interval should be < 10 minutes.

The number of Discordant 1st twin – 9 and 2nd twin - 26. Percentages of discordant twins were higher among 2nd twins and the loss was higher in the 2nd twin 12 babies Vs 2 of the 1st twin.

APGAR scores were lower in the 2nd twin in our study, hence the perinatal hypoxia and the mortality for the 2nd twin was higher.

Most of the deaths and morbidity were due to prematurity, and the loss of the 2nd twin was higher than the 1st twin mainly due to RDS, IUGR, and Birth asphyxia.

With respect to chorionicity, MC twins have a higher mortality due to the vascular anastamosis between the twins sharing a common placenta. Twin

pregnancies complicated by TTTS and single fetal death adds on to the mortality.

Even after excluding TTTS, mortality in MC twin pregnancies were higher than DC twin pregnancies. So, modern surveillance and intervention seems inefficient in MC twins compared with DC twins. It appears that third-trimester MC pregnancies at risk of perinatal death are difficult to identify. The most common cause of death in MC twins were prematurity, IUGR and sepsis. Discordancy was more with MC twins, 20 Vs 15 in DC twins with the loss being 10 Vs 4 in MC and DC twins. Longer delivery interval time between the babies affects MC twins more than DC twins.

Birth weight is approximately 100 grams higher in DC twins than in MC twins. The average length of NICU stay and the number of NICU admissions were higher in MC twins than in DC twins raising the mortality of MC twins to 177/1000 births Vs 92/1000 births in DC twins.

CONCLUSION

With the increased age of motherhood & with the increased number of mothers seeking for infertility treatments, Assisted reproductive technologies are been used widespread, leading onto the increased incidence of twin gestations. Any patient with a multiple gestation should be clinically managed as a high risk pregnancy.

The multidisciplinary team should be led by an obstetrician and should include midwives, Sonologist, Neonatologist and Anaesthetist. Such a service would provide a structured plan that will enable early detection, appropriate management and effective use of the resources for the antenatal, intrapartum and postnatal needs of the patients.

The conclusions derived from this clinical study are as follows:

Perinatal / Neonatal morbidity and mortality are significantly higher in multiple gestations than singleton pregnancies. Out of the perinatal mortality of the twins, the perinatal mortality of 2nd of the twin is higher than that of 1st twin due to various reasons. Outcome was poorer for MC twins than in DC twins. Also the chorionicity is important in assessing the perinatal outcome of the twins than the zygosity, which can only be determined using genetic testing.

Providing regular antenatal checkup to all mothers having multiple gestations will improve pregnancy outcome. Obstetricians can co-ordinate with dietician to direct efforts towards appropriate weight gain and adequate nutritional supplementation when necessary to achieve best feto-maternal outcome.

Ultrasound evaluation is the single most important diagnostic test in multiple gestations. Incidence of congenital malformations is two to three times higher in multiple gestations when compared to singleton pregnancies.
All patients with multiple gestations should have a thorough first and second trimester USG to assess chorionicity, amnionicity, individual fetal growth and congenital malformations.

Early detection of maternal complications and its management will improve outcome. The presentation of each fetus must be sonographically verified as soon as the patient with multiple pregnancy presents in labour.

Generally the mode of delivery should be vaginal when both twins present as vertex and caesarean section when 1st twin is non vertex. Active management of the second twin with an optimal interval of <10 minutes improves perinatal outcome of IInd of the twin. Immediate neonatal intensive care contributes to improved perinatal and neonatal outcome.

ANENCEPHALIC IST TWIN WITH FETUS PAPYRACEUS OF 2ND TWIN

FETUS PAPYRACEUS OF 2ND TWIN

ACARDIAC TWIN- AN EFFECT OF TRAP-AT 20 WEEKS OF GESTATION

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PROFORMA

Name:

Age:

IP.No:

DOA:

Do Deli:

DOD:

Gravida:

Para:

LMP:

Booked:

Menstrual History:

Marital History:

Obstetric History:

PRESENT HISTORY: I Trimester:

II Trimester:

III Trimester:

COMPLICATIONS DURING PREGNANCY:

1. Hyperemesis
2. UTI
3. APH
4. Anemia
5. PIH
6. Pressure Symptoms
7. Hydramnios

PAST HISTORY:

Drug intake : Clomiphene: Gonadotropins

OC Pills

Family History of twins:

Maternal

Paternal

EXAMINATION:

Investigations: Urine Albumin

Sugar

Deposits

Hemoglobin

Blood Sugar

USG

LABOUR:

1. Onset of labour spontaneous / induced
2. Period of gestation at which labour starts
3. Complications during labour

   PROM, Cord, prolapse,Atonic PPH

4. Presentation: Ist baby:

   IInd baby:

5. Mode of delivery of Ist baby:

   IInd baby:

6. Time interval:
7. Type of placenta:

BABIES:

1. Birth weight of Ist baby:

   IInd baby:

2. Discordancy: Ist baby:

   IInd baby:

3. 5 minute APGAR Ist baby:

   IInd baby:

4. Admission to NICU: **Yes/No**

   Ist baby:

IInd baby:

5. Duration of NICU stay:

Ist baby:

IInd baby:

6. Discharged alive: **Yes/No**

Ist baby:

IInd baby:

7. Diagnosis on discharge:

Ist baby:

IInd baby:

8. If expired, cause of death:

Ist baby:

IInd baby:

S.NO	NAME	AGE	PARITY	GA IN WEEKS	BOOKING STATUS	SPONTANEOUS LABOUR	INDUCED MOTHER SAKE	MATERNAL COMPLICATIONS	CHORIONICITY	TIME INTERVAL	DISCORDANT	PRESENTATION		MOD		B.WT KGS		APGAR		NICU ADMISSIONS		DAYS OF NICU STAY		DIAGNOSIS AT DISCHARGE		EXPIRED		CAUSE OF DEATH	
										IN MINUTES		TWIN 1	TWIN 2	TWIN 1	TWIN 2	TWIN 1	TWIN 2	TWIN 1	TWIN 2	TWIN 1	TWIN 2	TWIN 1	TWIN 2	TWIN 1	TWIN 2	TWIN 1	TWIN 2	TWIN 1	TWIN 2
1	BHUVANESHWARI	22	PRIMI	35	YES	YES	-	-	1	13	-	V	V	LN	LN	2	2.1	I	I	-	-	-	-	-	-	-	-	-	-
2	SHARADHA	27	G3P2L2	38	YES	YES	-	-	1	10	-	V	V	LN	LN	2.8	2.5	I	I	-	-	-	-	-	-	-	-	-	-
3	JEEVA	27	PRIMI	34	YES	YES	-	-	1	3	-	V	V	LN	LN	1.2	1.3	I	II	YES	YES	8	10	1	1	-	-	-	-
4	SELVI	23	PRIMI	37	YES	YES	-	-	1	20	YES	V	B	LN	ABD	2	1.5	I	II	-	YES	-	10	-	1	-	-	-	-
5	KAVITHA	31	G3P2L0	34	YES	YES	-	1	1	2	-	B	B	EMER LSCS		1.6	1.6	II	II	YES	YES	8	8	1	1	-	-	-	-
6	NATHIYA	21	PRIMI	37	YES	YES	-	1	1	7	-	V	B	LN	ABD	2.2	2	I	I	-	-	-	-	-	-	-	-	-	-
7	KALAIVANI	28	PRIMI	37	YES	YES	-	5	1	2	-	V	T	EMER LSCS		2.4	2.3	II	I	YES	-	5	-	NO	-	YES	-	8	-
8	DHANALAXMI	28	G3P1L1A1	37	YES	YES	-	-	1	5	-	B	B	EMER LSCS		2.4	2.3	II	II	YES	YES	2	2	1	1	-	-	-	-
9	TAMILARASI	23	PRIMI	33	YES	-	YES	1	1	5	-	B	V	ABD	LN	1.1	1.1	III	III	YES	YES	15	15	NO	NO	YES	YES	7	7
10	AMBIKA	25	G3P2L1	40	NO	YES	-	-	1	4	-	V	V	LN	LN	2.2	2	I	I	-	-	-	-	-	-	-	-	-	-
11	SELVI	28	G2P1L1	34	YES	YES	-	3	1	2		B	B	EMER LSCS		1.7	2	I	I	-	-	-	-	-	-	-	-	-	-
12	VANAROJA	28	PRIMI	39	YES	YES	-	1	1	5	-	B	V	EMER LSCS		2	2.4	I	I	-	-	-	-	-	-	-	-	-	-
13	KALAISELVI	22	G2P1L1	37	YES	YES	-	-	1	10	-	V	B	LN	ABD	2.3	2	I	II	-	YES	-	13	-	1	-	-	-	-
14	SHANTHI	21	PRIMI	34	YES	YES	-	-	1	20	-	V	V	LN	LN	1.8	1.6	II	II	YES	YES	15	17	1	1	-	-	-	-
15	KAVERI	27	G3P2L2	38	YES	YES	-	-	1	6	-	V	B	LN	ABD	2.2	2	I	I	-	-	-	-	-	-	-	-	-	-
16	UMAMAHESWARI	22	PRIMI	36	YES	YES	-	1	1	5	-	B	V	ABD	LN	2.3	2	II	II	YES	YES	4	4	1	1	-	-	-	-
17	GOWTHAMI	31	G3P1L2A1	39	YES	YES	-	-	1	23	-	V	V	LN	LN	2.7	2.6	I	I	-	-	-	-	-	-	-	-	-	-
18	VASANTHI	19	PRIMI	35	YES	YES	-	-	1	5	-	V	B	LN	ABD	1.7	1.8	I	I	YES	YES	11	11	5	5	-	-	-	-
19	MUTHULAXMI	27	G2P1L1	36	NO	YES	-	-	1	62	YES	V	V	LN	LN	2.6	2	I	I	-	-	-	-	-	-	-	-	-	-
20	MALATHI	22	G2P1L1	32	YES	YES	-	-	1	10	-	V	V	LN	LN	1.5	1.2	II	III	YES	YES	3	4	NO	NO	YES	YES	12	12
21	MANGALAM	28	G2P1L1	38	YES	YES	-	-	1	2	-	B	V	EMER LSCS		2.4	2.2	I	I	-	-	-	-	-	-	-	-	-	-
22	PARAMESHWARI	23	PRIMI	35	YES	YES	-	-	1	2	-	V	B	LN	ABD	2.6	2.4	I	I	-	-	-	-	-	-	-	-	-	-
23	RANJITHA	20	PRIMI	38	YES	YES	-	-	1	7	-	B	B	ABD	ABD	1.9	1.8	I	I	-	-	-	-	-	-	-	-	-	-
24	SHYAMALA DEVI	31	G2P1L1	38	YES	YES	-	-	1	11	-	V	V	LN	LN	2.7	2.5	I	I	-	-	-	-	-	-	-	-	-	-
25	REHMANIYA	30	PRIMI	32	NO	YES	-	1,5	1	1	YES	T	B	EMER LSCS		1.9	1.3	IV	II	-	YES	-	1	-	NO	YES	YES	9	7
26	MANGAYAR KARASI	29	PRIMI	30	YES	YES	-	1	1	15	-	V	V	LN	LN	1.4	1.3	I	II	-	YES	-	10	-	1	-	-	-	-
27	BANUPRIYA	20	PRIMI	36	YES	YES	-	-	1	7	-	V	V	LN	LN	1.9	2.2	I	I	-	-	-	-	-	-	-	-	-	-
28	DEVIKA	23	G4P3L3	36	YES	YES	-	-	1	30	-	V	V	LN	LN	2.6	2.1	I	I	-	-	-	-	-	-	-	-	-	-
29	KAYALVIZHI	29	G2P1L1	37	YES	YES	-	1,5	1	2	-	B	V	ELECTIVE LSCS		2.5	3	I	I	-	-	-	-	-	-	-	-	-	-
30	RENUKA	23	PRIMI	39	YES	YES	-	1	1	5	-	V	V	LN	LN	2.1	2.6	I	I	-	-	-	-	-	-	-	-	-	-
31	KAVITHA	26	PRIMI	35	NO	YES	-	1	1	13	-	V	V	LN	LN	2	1.8	I	I	-	-	-	-	-	-	-	-	-	-
32	NITHYA	27	PRIMI	35	YES	YES	-	-	1	4	-	V	V	LN	LN	1.7	1.6	I	II	YES	YES	3	3	1	1	-	-	-	-
33	SARASWATHY	24	G2P1L1	36	YES	YES	-	-	1	15	-	V	V	LN	LN	2.5	2.6	I	I	-	-	-	-	-	-	-	-	-	-
34	SUDHA	27	G2P1L1	36	YES	YES	-	1	1	4	-	V	V	LN	LN	1.75	1.5	I	I	-	-	-	-	-	-	-	-	-	-
35	AMUDHA	23	PRIMI	36	YES	-	YES	2,4	1	1	-	V	V	EMER LSCS		1.6	1.9	I	I	-	-	-	-	-	-	-	-	-	-
36	VENNILA	20	PRIMI	40	YES	YES	-	-	1	6	-	V	V	LN	LN	1.4	1.6	III	II	YES	YES	3	3	NO	1	YES	-	7	-
37	AMBIKA	28	PRIMI	38	YES	YES	-	-	1	5	-	V	V	LN	LN	2	1.9	I	I	-	-	-	-	-	-	-	-	-	-
38	NADHIYA	24	G2P1L1	36	YES	YES	-	-	1	14	-	V	B	LN	ABD	2	1.5	I	II	-	YES	-	2	-	NO	-	YES	-	12
39	AMUDHARANI	25	PRIMI	37	YES	YES	-	-	1	21	-	V	B	LN	ABD	2.5	2	I	I	-	-	-	-	-	-	-	-	-	-
40	NAVAMANI	23	PRIMI	38	NO	YES	-	-	1	3	-	B	B	EMER LSCS		2.4	2	I	I	-	-	-	-	-	-	-	-	-	-
41	KALAIMANI	26	G2P1L1	36	NO	YES	-	-	1	2	-	V	V	LN	LN	2	2	I	I	-	-	-	-	-	-	-	-	-	-
42	CHITRA	27	G2P1L1	35	YES	YES	-	-	1	5	-	V	V	LN	LN	2	1.7	I	I	-	-	-	-	-	-	-	-	-	-
43	PRIYA	27	PRIMI	34	YES	YES	-	-	1	8	-	B	B	ABD	ABD	2	2	II	II	YES	YES	8	10	1	1	-	-	-	-
44	MALLIGA	22	PRIMI	34	YES	YES	-	-	1	1	-	V	V	EMER LSCS		2	1.9	I	I	YES	YES	3	3	1	1	-	-	-	-
45	RAMANI	32	G3	32	YES	YES	-	-	1	60	YES	V	B	LN	ABD	1.8	1	I	III	-	YES	-	3	-	NO	-	YES	-	10

46	KALAISELVI	30	G4P3L3	34	YES	YES	-	-	1	11	-	V	B	LN	ABD	2	1.7	I	III	YES	YES	2	3	4	NO	-	YES	-	7
47	RAGINI	28	G2P1L1	37	NO	YES	-	-	1	2	-	V	V	LN	LN	2.2	2.3	I	I	-	-	-	-	-	-	-	-	-	-
48	USHA	27	G2P1L1	39	NO	YES	-	-	1	35	-	V	V	LN	LN	2	2.1	I	I	-	-	-	-	-	-	-	-	-	-
49	ILLAVARASI	24	PRIMI	37	YES	YES	-	-	1	2	-	B	B	EMER LSCS		2	1.8	I	II	YES	YES	3	5	1	1	-	-	-	-
50	NADHIYA	24	G2P1L1	34	YES	YES	-	-	1	27	-	V	V	LN	LN	2	1.7	I	I	-	-	-	-	-	-	-	-	-	-
51	ADITHYA	30	G3	37	YES	YES	-	1	1	3	-	B	V	ABD	LN	2	2.2	I	I	-	-	-	-	-	-	-	-	-	-
52	AMUDHARANI	20	PRIMI	35	YES	-	YES	1	1	5	YES	V	B	LN	ABD	2.5	1.6	IV	II	-	YES	-	23	-	1	YES	-	13	-
53	RADHIKA	22	PRIMI	33	YES	YES	-	-	1	5	YES	V	B	LN	ABD	1	1.5	II	II	YES	YES	5	5	10	10	-	-	-	-
54	RAJATHI	25	G2P1L1	36	YES	-	YES	1	1	10	YES	V	V	LN	LN	2.6	2	II	II	YES	YES	2	8	1	1	-	-	-	-
55	AJITHA	27	PRIMI	37	YES	-	YES	1	1	8	-	V	V	O	LN	1.9	2	I	I	-	-	-	-	-	-	-	-	-	-
56	MALATHI	22	G2P1L1	38	NO	YES	-	-	1	2	-	V	V	LN	LN	2.5	2.6	I	I	-	-	-	-	-	-	-	-	-	-
57	MARIAMMAL	21	PRIMI	34	NO	YES	-	-	1	5	-	V	V	LN	LN	1.6	1.7	I	II	YES	YES	10	14	1	1	-	-	-	-
58	MARIKANU	20	PRIMI	34	YES	YES	-	-	1	5	-	V	V	LN	LN	2.4	2.3	III	II	YES	YES	18	18	NO	1	YES	-	7	-
59	UMA	30	PRIMI	37	YES	YES	-	-	1	5	-	B	B	ELECTIVE LSCS		2	2	I	I	-	-	-	-	-	-	-	-	-	-
60	SUDHA	23	PRIMI	39	YES	YES	-	1	1	2	-	V	V	EMER LSCS		2.5	2.3	I	I	-	-	-	-	-	-	-	-	-	-
61	KAVITHA	25	PRIMI	39	YES	YES	-	-	1	10	-	V	V	LN	LN	1.7	1.5	I	II	YES	YES	1	6	1	1	-	-	-	-
62	DHANAKODI	27	G3	34	NO	YES	-	1,4	1	2	-	B	B	EMER LSCS		1.9	2	I	II	-	YES	-	5	-	1	-	-	-	-
63	SURYAKALA	25	G2P1L1	40	YES	YES	-	-	1	10	-	V	V	LN	LN	2.2	2.2	I	I	-	-	-	-	-	-	-	-	-	-
64	MEGALA	25	G2P1L1	37	YES	YES	-	-	1	10	-	V	V	LN	LN	1.7	1.8	I	I	-	-	-	-	-	-	-	-	-	-
65	SANGEETHA	26	PRIMI	34	YES	-	YES	1	1	10	YES	V	V	LN	LN	1.3	1	II	III	YES	YES	24	26	2	NO	-	YES	-	2
66	VIJAYALAKSHMI	23	PRIMI	38	YES	YES	-	-	1	8	-	V	B	LN	ABD	2.4	2	I	I	-	-	-	-	-	-	-	-	-	-
67	SAGAYARANI	24	G2P1L1	37	YES	YES	-	4,5	1	60	YES	V	T	LN	IPV	1.6	2.3	I	I	-	-	-	-	-	-	-	-	-	-
68	NAGALAKSHMI	30	G3	38	YES	YES	-	1	1	1	-	T	T	EMER LSCS		2.9	2.5	I	I	-	-	-	-	-	-	-	-	-	-
69	DHANA	28	PRIMI	40	NO	YES	-	-	1	5	-	B	B	ELECTIVE LSCS		2.6	2.5	I	I	-	-	-	-	-	-	-	-	-	-
70	SANGEETHA	21	PRIMI	32	YES	YES	-	-	1	3	-	B	V	ABD	LN	1.25	1.5	III	III	YES	YES	15	12	NO	NO	YES	YES	7	2
71	SUDHARANI	21	PRIMI	38	YES	YES	-	2	1	16	-	V	V	LN	LN	2.2	2	I	I	-	-	-	-	-	-	-	-	-	-
72	AMUDHA	28	G2P1L1	40	YES	YES	-	2,5	1	1	-	V	V	EMER LSCS		2.7	2.7	I	I	-	-	-	-	-	-	-	-	-	-
73	LEEMA	23	PRIMI	39	YES	YES	-	4,5	1	12	-	V	V	LN	LN	2	2	I	I	-	-	-	-	-	-	-	-	-	-
74	SATHYA	23	G3	36	NO	YES	-	1	1	15	-	V	B	LN	ABD	2.5	2.25	I	II	YES	YES	3	5	4	4	-	-	-	-
75	ANUSHIYA	22	PRIMI	36	YES	YES	-	1	1	2	-	B	V	EMER LSCS		1.9	2.1	I	I	-	-	-	-	-	-	-	-	-	-
76	RADHA	26	PRIMI	39	YES	YES	-	1	1	6	-	V	V	LN	LN	2.1	2	I	I	-	-	-	-	-	-	-	-	-	-
77	RAMANI	27	G2P1L1	34	YES	YES	-	1	1	7	-	V	V	LN	LN	2	2	I	I	-	-	-	-	-	-	-	-	-	-
78	MALAR	25	PRIMI	36	YES	YES	-	1	1	2	-	T	V	EMER LSCS		1.8	1.9	I	I	-	-	-	-	-	-	-	-	-	-
79	PATTUROJA	25	G2P1L1	36	YES	YES	-	-	1	5	-	V	V	LN	LN	1.9	1.9	I	II	YES	YES	7	14	1	1	-	-	-	-
80	REETA	23	PRIMI	37	YES	YES	-	1	1	31	-	V	V	LN	LN	2	2	I	I	-	-	-	-	-	-	-	-	-	-
81	NASREEN	22	PRIMI	36	NO	YES	-	-	1	5	-	V	V	LN	LN	2.3	2	I	I	YES	YES	7	7	4	4	-	-	-	-
82	REVATY	22	PRIMI	39	YES	-	YES	1	1	1	-	V	V	EMER LSCS		2.6	2.1	I	I	YES	YES	7	7	4	4	-	-	-	-
83	ILAVARASI	21	PRIMI	38	YES	YES	-	1,4	1	5	-	V	B	LN	ABD	2	1.8	I	II	-	YES	-	3	-	1	-	-	-	-
84	LAKSHMI	30	PRIMI	36	NO	YES	-	1,4	1	9	-	V	V	LN	LN	1.8	1.7	I	II	YES	YES	6	10	1	1	-	-	-	-
85	SUDHA	30	G2P1L1	35	YES	-	YES	2	1	3	-	V	B	LN	ABD	2	1.9	I	II	-	YES	-	3	-	6	-	-	-	-
86	SUGUNA	24	PRIMI	36	YES	YES	-	-	1	28	-	V	V	LN	LN	2.4	2.4	I	I	-	-	-	-	-	-	-	-	-	-
87	ABINAYA	28	G2P1L1	36	YES	YES	-	-	1	60	-	V	C	LN	EM LSCS	2.6	2.9	I	III	-	YES	-	1	-	NO	-	YES	-	9
88	ILAVARASI	22	PRIMI	38	NO	YES	-	1	1	5	-	V	B	LN	ABD	2	1.8	I	I	-	YES	-	6	-	1	-	-	-	-
89	GOWRI	26	PRIMI	37	YES	YES	-	-	1	16	-	V	B	LN	ABD	2.5	2.1	I	II	YES	YES	5	8	1	1	-	-	-	-
90	MAHALAXMI	23	G3	38	YES	YES	-	-	1	8	YES	B	B	ABD	ABD	2.3	1.5	I	I	-	YES	-	10	-	4	-	-	-	-
91	BARGAVI	25	PRIMI	40	YES	-	YES	2	1	1	YES	V	V	EMER LSCS		2.8	2.2	I	II	-	YES	-	2	-	1	-	-	-	-
92	CHITRA	36	G3	36	YES	YES	-	-	1	6	YES	V	V	LN	LN	2.6	2	I	I	-	-	-	-	-	-	-	-	-	-

93	SUSEELA	27	PRIMI	37	YES	YES	-	1	1	2	-	V	V	EMER LSCS		2	1.9	III	I	YES	YES	2	5	NO	1	YES	-	2	-
94	SUMITHRA	20	PRIMI	35	NO	-	YES	3	1	2	-	V	V	EMER LSCS		2.2	2.2	I	I	YES	YES	3	3	6	6	-	-	-	-
95	KAMALAM	23	PRIMI	35	YES	YES	-	-	1	5	-	V	V	LN	LN	1.7	1.7	I	II	YES	YES	6	6	4	4	-	-	-	-
96	SANGEETHA	23	PRIMI	36	YES	YES	-	1	1	25	-	V	V	LN	LN	1.5	1.5	I	I	YES	YES	7	7	1	1	-	-	-	-
97	RATHIDEVI	27	G2P1L1	37	YES	YES	-	-	1	10	-	V	V	LN	LN	2.8	2.4	I	I	-	-	-	-	-	-	-	-	-	-
98	LAKSHMI	28	PRIMI	37	YES	YES	-	-	1	12	-	V	V	LN	LN	2.5	2.4	I	I	-	-	-	-	-	-	-	-	-	-
99	KALAIYARASI	26	G2P1L1	35	YES	YES	-	-	1	12	YES	V	V	LN	LN	1.6	2.2	I	I	YES	YES	5	7	1	1	-	-	-	-
100	SUDHA	23	G3	35	YES	YES	-	1	1	45	-	V	V	LN	LN	2	1.9	I	II	-	YES	-	14	-	2	-	-	-	-
101	JANCYMARY	24	PRIMI	38	YES	YES	-	1	1	9	-	V	V	LN	LN	2.7	2.3	I	I	-	-	-	-	-	-	-	-	-	-
102	KANAMAL	22	G2P1L1	38	YES	YES	-	-	1	12	YES	V	V	LN	LN	2.5	1.9	I	I	-	-	-	-	-	-	-	-	-	-
103	ARCHANA	24	PRIMI	37	YES	YES	-	-	1	2	-	V	V	LN	LN	2	2.1	I	I	-	-	-	-	-	-	-	-	-	-
104	BAVYA	24	PRIMI	37	YES	YES	-	-	1	5	-	V	V	LN	LN	1.8	1.9	I	I	-	-	-	-	-	-	-	-	-	-
105	CHARU	22	PRIMI	37	YES	YES	-	-	1	5	-	V	V	LN	LN	2.1	2.1	I	I	-	-	-	-	-	-	-	-	-	-
106	DEVI	32	G3	36	YES	YES	-	-	1	5	-	V	V	LN	LN	2	1.9	I	I	YES	YES	3	3	4	4	-	-	-	-
107	ELIZABETH	30	G2P1L1	30	YES	YES	-	-	1	3	-	B	B	ABD	ABD	1.1	1	II	III	YES	YES	6	2	NO	NO	YES	YES	2	2
108	FATHIMA	28	G2P1L1	36	YES	YES	-	-	1	3	-	V	B	LN	ABD	1.8	1.5	I	I	-	-	-	-	-	-	-	-	-	-
109	GAYATHRI	30	G2P1L1	36	YES	YES	-	-	1	6	-	B	B	ABD	ABD	1.6	1.7	I	I	-	-	-	-	-	-	-	-	-	-
110	HARINI	22	G2P1L1	34	YES	YES	-	-	1	4	-	V	V	LN	LN	1.9	1.6	I	II	-	YES	-	12	-	1	-	-	-	-
111	INDRANI	28	PRIMI	38	YES	YES	-	-	1	4	-	V	V	LN	LN	2.8	2.7	I	II	-	YES	-	14	-	6	-	-	-	-
112	JENIFER	23	PRIMI	35	YES	YES	-	-	1	4	-	V	V	LN	LN	2.3	2.1	I	I	-	-	-	-	-	-	-	-	-	-
113	KALA	22	PRIMI	36	YES	YES	-	-	1	6	YES	V	V	LN	LN	2.3	1.5	I	III	-	YES	-	30	-	NO	-	YES	-	12
114	LAKSHMI	32	PRIMI	38	NO	YES	-	-	1	4	-	V	V	LN	LN	3.2	3	I	I	-	-	-	-	-	-	-	-	-	-
115	NIRMALA	22	PRIMI	32	YES	YES	-	1	2	20	YES	V	V	LN	LN	0.8	1.16	IV	III	-	YES	-	1	-	NO	YES	YES	13	7
116	TAMILARASI	30	PRIMI	35	YES	YES	-	-	2	5	-	V	V	LN	LN	1.8	1.6	I	II	-	YES	-	5	-	1	-	-	-	-
117	LAKSHMI	40	G4P3L3	32	YES	YES	-	-	2	2	-	V	V	LN	LN	1.2	1	II	III	YES	YES	6	4	3	NO	-	YES	-	2
118	ARIYANATCHI	23	PRIMI	31	YES	-	YES	1	2	2	-	B	V	ABD	LN	0.8	0.8	III	III	YES	YES	1	1	NO	NO	YES	YES	2	2
119	MAHESWARI	20	PRIMI	32	YES	YES	-	-	2	5	-	V	B	LN	ABD	1.5	1.5	II	II	YES	YES	7	11	10	10	-	-	-	-
120	SARAYA	23	G2P1L1	38	NO	YES	-	1,5	2	10	-	B	V	ABD	LN	2.2	2.7	I	I	-	-	-	-	-	-	-	-	-	-
121	SELVI	32	G2P1L1	36	YES	YES	-	-	2	2	-	V	V	LN	LN	2	2	I	I	-	-	-	-	-	-	-	-	-	-
122	SANGEETHA	25	G2P1L1	37	YES	YES	-	-	2	15	YES	V	B	LN	ABD	2.5	2	I	II	-	YES	-	7	-	1	-	-	-	-
123	UMARANI	20	PRIMI	37	YES	YES	-	4	2	2	-	V	B	EMER LSCS		1.1	1.2	II	II	YES	YES	5	5	3	3	-	-	-	-
124	CHITRA	25	G3	38	NO	YES	-	-	2	15	-	B	V	ABD	LN	2.4	2.5	I	I	-	-	-	-	-	-	-	-	-	-
125	TAMILSELVI	26	G2P1L1	37	YES	YES	-	-	2	7	YES	V	B	LN	ABD	2.3	1.7	I	I	-	-	-	-	-	-	-	-	-	-
126	JEYANTHI	31	G2P1L1	38	NO	YES	-	5	2	2	-	V	V	EMER LSCS		3.3	2.7	I	I	-	-	-	-	-	-	-	-	-	-
127	SUDHA	29	G2P1L1	32	YES	YES	-	-	2	4	-	V	V	LN	LN	1.2	1.5	II	II	YES	YES	7	7	1	1	-	-	-	-
128	NADHIYA	23	G2P1L1	38	NO	YES	-	4	2	5	YES	T	V	EMER LSCS		2	2.7	I	I	-	-	-	-	-	-	-	-	-	-
129	RAJESWARI	26	G3	37	YES	YES	-	-	2	4	-	B	V	ABD	LN	1.9	2.5	I	I	-	-	-	-	-	-	-	-	-	-
130	LALLI	30	G3	34	YES	YES	-	-	2	2	-	V	V	LN	LN	1.8	1.6	I	I	-	-	-	-	-	-	-	-	-	-
131	SHANTHI	27	PRIMI	37	YES	YES	-	4	2	5	-	V	V	LN	LN	2	2	I	I	-	-	-	-	-	-	-	-	-	-
132	MALATHY	24	PRIMI	36	YES	YES	-	1,4	2	3	-	V	V	EMER LSCS		1.8	1.4	I	II	YES	YES	6	10	1	1	-	-	-	-
133	UMAMAHESWARI	27	G2P1L1	35	YES	YES	-	1	2	26	-	V	B	LN	ABD	2	2.2	I	I	-	-	-	-	-	-	-	-	-	-
134	SELVI	23	PRIMI	37	YES	YES	-	1	2	20	-	V	V	LN	LN	2.5	2	I	I	-	-	-	-	-	-	-	-	-	-
135	SIVASAKTHI	25	G2P1L1	38	NO	YES	-	-	2	8	-	V	V	LN	LN	2.6	2.5	I	I	-	-	-	-	-	-	-	-	-	-
136	SANGEETHA	22	PRIMI	37	YES	YES	-	1,5	2	1	-	T	V	EMER LSCS		2	1.9	IV	I	-	-	-	-	NO	-	YES	-	9	-
137	SATHYA	23	G2P1L1	38	NO	YES	-	1	2	2	YES	V	V	EMER LSCS		3.1	2.1	II	III	YES	YES	5	2	1	NO	-	YES	-	7
138	SANTHASEELA	26	PRIMI	37	YES	YES	-	-	2	11	-	V	V	LN	LN	2	1.8	II	II	YES	YES	3	3	1	1	-	-	-	-
139	RADHIKA	22	G3	40	NO	YES	-	-	2	5	-	B	V	ABD	LN	2.5	2.7	I	I	-	-	-	-	-	-	-	-	-	-

140	SUGANTHI	25	PRIMI	40	NO	YES	-	-	2	4	-	V	B	LN	ABD	2	1.7	I	II	-	YES	-	7	-	3	-	-	-	-
141	JEYA	24	G2P1L1	34	YES	YES	-	-	2	5	-	V	V	LN	LN	1.4	1.4	II	II	YES	YES	5	5	3	3	-	-	-	-
142	MUTHAMILSELVI	25	G2P1L1	38	NO	YES	-	2	2	5	YES	V	V	LN	LN	2.5	2	I	I	-	-	-	-	-	-	-	-	-	-
143	UMA	30	PRIMI	40	NO	YES	-	-	2	4	-	B	V	EMER LSCS		2.1	2.2	I	I	-	-	-	-	-	-	-	-	-	-
144	INDUMATHY	20	PRIMI	38	NO	YES	-	-	2	2	-	T	B	ELECTIVE LSCS		2.2	2	I	I	-	-	-	-	-	-	-	-	-	-
145	MUTHULAXMI	27	G2P1L1	30	YES	YES	-	-	2	4	-	B	V	ABD	LN	1	1.1	III	III	YES	YES	2	1	NO	NO	YES	YES	10	10
146	THENMOZHI	28	G2P1L1	38	NO	YES	-	1	2	4	-	V	V	LN	LN	2.5	2.5	I	I	-	-	-	-	-	-	-	-	-	-
147	TAMILSELVI	20	PRIMI	34	YES	YES	-	-	2	6	-	V	V	LN	LN	1.6	1.2	I	II	-	YES	-	11	-	1	-	-	-	-
148	LAKSHMI	28	G2P1L1	34	YES	-	YES	1	2	4	-	B	B	EMER LSCS		1.9	1.6	II	III	YES	YES	5	1	6	NO	-	YES	-	7
149	RAJAKUMARI	26	PRIMI	37	YES	YES	-	1	2	3	-	B	V	ABD	LN	1.9	2.1	I	I	-	-	-	-	-	-	-	-	-	-
150	UMARANI	23	G2P1L1	36	YES	YES	-	1	2	14	YES	V	V	LN	LN	1.2	1.9	II	I	YES	-	16	-	NO	-	YES	-	2	-
151	SENTHAMARAI	29	G3	39	NO	YES	-	-	2	15	YES	V	B	LN	ABD	2.3	3.1	I	I	-	-	-	-	-	-	-	-	-	-
152	SUMATHY	28	G2P1L1	36	YES	YES	-	-	2	1	-	B	V	EMER LSCS		2.2	2.1	I	I	-	-	-	-	-	-	-	-	-	-
153	PALANIYAMAL	21	PRIMI	38	NO	YES	-	1	2	7	-	V	V	LN	LN	2.2	1.8	I	II	-	YES	-	2	-	3	-	-	-	-
154	SUVITHA	26	G2P1L1	36	YES	YES	-	-	2	12	-	V	V	LN	LN	2	2	I	I	-	-	-	-	-	-	-	-	-	-
155	MALA	29	PRIMI	32	YES	YES	-	-	2	1	-	B	B	EMER LSCS		2.1	1.7	I	II	-	YES	-	13	-	1	-	-	-	-
156	KALAIYARASI	25	PRIMI	36	YES	YES	-	5	2	10	-	V	V	LN	LN	1.7	1.7	II	II	YES	YES	6	6	1	1	-	-	-	-
157	RAMANI	25	PRIMI	29	NO	-	YES	2	2	8	YES	V	V	LN	LN	1.5	0.8	III	IV	YES	-	3	-	NO	NO	YES	YES	11	11
158	GUNASUNDARI	27	G2P1L1	32	YES	YES	-	1	2	48	-	V	V	LN	LN	1.5	1.2	II	II	YES	YES	5	20	1	1	-	-	-	-
159	RADHIKA	30	PRIMI	32	YES	YES	-	-	2	180	-	V	T	LN	IPV	1.2	1	II	IV	YES	-	10	-	NO	NO	YES	YES	2	7
160	RASIYABEGAM	23	PRIMI	38	NO	YES	-	-	2	1	-	T	T	EMER LSCS		2.6	2.4	I	II	-	YES	-	2	-	1	-	-	-	-
161	PREMAVATHY	26	G2P1L1	35	YES	YES	-	2	2	11	-	V	V	LN	LN	2.4	2	II	I	YES	-	15	-	NO	-	YES	-	8	-
162	REKHA	23	G2P1L1	37	YES	YES	-	-	2	1	-	V	V	EMER LSCS		1.8	1.5	II	II	YES	YES	3	5	1	1	-	-	-	-
163	MALA	29	G2P1L1	30	YES	YES	-	-	2	7	-	B	V	ABD	LN	1.5	1.5	II	II	YES	YES	3	7	1	1	-	-	-	-
164	VIJAYA	28	PRIMI	39	NO	YES	-	-	2	15	YES	V	V	LN	LN	2.6	1.4	II	III	YES	YES	5	2	1	NO	-	YES	-	2
165	MAHALAXMI	25	G5	30	YES	YES	-	-	2	4	-	V	V	LN	LN	1.5	1.6	II	II	YES	YES	10	10	4	4	-	-	-	-
166	SHANMUGAPRIYA	25	G2P1L1	38	NO	YES	-	-	2	5	-	V	V	LN	LN	3.3	3.2	I	I	-	-	-	-	-	-	-	-	-	-
167	SANTHASEELA	29	G2P1L1	37	YES	YES	-	-	2	1	YES	V	V	ELECTIVE LSCS		2.2	1.5	I	II	-	YES	-	15	-	1	-	-	-	-
168	PRAVEENA	25	PRIMI	38	NO	YES	-	-	2	6	-	V	V	LN	LN	2.2	2.1	I	I	-	-	-	-	-	-	-	-	-	-
169	THILAKAVATHI	30	G4P3L3	38	NO	YES	-	4	2	5	YES	V	V	LN	LN	1.7	2.4	I	I	-	-	-	-	-	-	-	-	-	-
170	RENUKA	22	PRIMI	38	NO	YES	-	-	2	2	YES	B	V	EMER LSCS		3.3	2	I	I	-	-	-	-	-	-	-	-	-	-
171	MALLIGA	32	G2P1L1	30	YES	-	YES	2	2	10	-	V	V	LN	LN	0.8	0.8	IV	III	-	YES	-	<1	NO	NO	YES	YES	3	3
172	BABY	25	G2P1L1	38	NO	YES	-	5	2	4	-	V	V	LN	LN	2.5	2.2	I	I	-	-	-	-	-	-	-	-	-	-
173	RADHIKA	30	PRIMI	32	YES	YES	-	2	2	180	-	V	T	LN	IPV	1	1	III	IV	YES	-	1	-	NO	NO	YES	YES	3	3
174	SUMATHY	23	G3	28	YES	YES	-	-	2	7	-	V	V	LN	LN	0.8	0.9	IV	III	-	YES	-	<1	NO	NO	YES	YES	10	10
175	NIROSHA	23	PRIMI	36	YES	YES	-	-	2	2	-	V	V	LN	LN	1.7	1.9	II	II	YES	YES	6	6	1	1	-	-	-	-
176	PRIYA	33	PRIMI	37	NO	YES	-	-	2	2	YES	V	V	LN	LN	2.5	1.2	I	II	-	YES	-	8	-	1	-	-	-	-

177	SARASWATHY	35	PRIMI	34	YES	YES	-	-	2	9	-	V	V	LN	LN	1.5	1.5	II	III	YES	YES	2	2	NO	NO	YES	YES	7	7
178	KASTURI	24	PRIMI	34	YES	YES	-	1	2	7	-	V	V	O	LN	2	1.5	II	III	YES	YES	10	5	1	NO	-	YES	-	2
179	MAHESWARI	25	PRIMI	37	NO	YES	-	2	2	6	-	V	B	LN	ABD	2.5	2.8	I	I	-	-	-	-	-	-	-	-	-	-
180	SUKANYA	21	PRIMI	35	YES	YES	-	-	2	2	-	B	B	EMER LSCS		2.7	2.3	I	I	-	-	-	-	-	-	-	-	-	-
181	VIJAYA	29	G5	34	YES	YES	-	1,4	2	10	YES	V	V	LN	LN	1.6	2.2	II	II	YES	YES	7	7	4	4	-	-	-	-
182	ANNAKODI	26	G4P3L3	37	NO	YES	-	1	2	3	-	V	V	LN	LN	2	2	I	I	-	-	-	-	-	-	-	-	-	-
183	SUREKHA	31	G2P1L1	36	YES	YES	-	-	2	9	-	V	V	LN	LN	2.8	2.2	I	I	-	-	-	-	-	-	-	-	-	-
184	ANJAMMAL	26	G3	34	YES	YES	-	-	2	26	-	V	V	LN	LN	2.2	2.2	I	I	-	-	-	-	-	-	-	-	-	-
185	ARTHI	20	PRIMI	34	YES	YES	-	-	2	2	-	V	V	LN	LN	1.4	1.2	II	II	YES	YES	10	14	3	3	-	-	-	-
186	BINDHU	20	PRIMI	32	YES	YES	-	-	2	2	-	B	B	ABD	ABD	1.2	1.2	II	II	YES	YES	14	16	3	3	-	-	-	-
187	CLARY	20	PRIMI	34	YES	YES	-	-	2	16	-	V	B	LN	ABD	1.6	1.2	I	II	-	YES	-	10	-	1	-	-	-	-
188	DANUSHYA	30	G2P1L1	32	YES	YES	-	-	2	12	-	V	V	LN	LN	1.2	1.1	II	II	YES	YES	10	20	10	10	-	-	-	-
189	ELCY	30	G2P1L1	33	YES	YES	-	-	2	1	YES	B	B	EMER LSCS		1.7	1.3	I	II	-	YES	-	19	-	1	-	-	-	-
190	FATHIMA	28	PRIMI	32	YES	YES	-	-	2	10	-	V	B	LN	ABD	1.6	1.8	II	I	YES	-	3	-	1	-	-	-	-	-
191	GANGA	32	G2P1L1	37	NO	YES	-	1	2	1	YES	V	V	EMER LSCS		2.6	1.8	I	II	-	YES	-	24	-	NO	-	YES	-	3
192	SANGAVI	23	PRIMI	32	YES	YES	-	-	2	1	-	V	V	LN	LN	1.6	1.5	II	II	YES	YES	14	16	10	10	-	-	-	-
193	MAHAPRIYA	23	PRIMI	38	NO	YES	-	-	2	15	YES	V	V	LN	LN	2.8	1.5	I	III	-	YES	-	12	-	NO	-	YES	-	3
194	DANALAKSHMI	27	PRIMI	32	YES	YES	-	-	3	5	YES	V	B	EMER LSCS		1.6	1.1	I	III	YES	YES	3	14	1	NO	-	YES	-	2
195	SUBHA	22	G2P1L1	30	YES	YES	-	-	3	5	-	V	V	LN	LN	1	1	III	III	YES	YES	1	1	NO	NO	YES	YES	8	7
196	RADHIKA	25	G2P1L1	36	NO	YES	-	-	3	9	YES	V	V	LN	LN	2.4	1.8	I	III	-	YES	-	5	-	NO	-	YES	-	7
197	CHITRA	34	G3	30	YES	YES	-	-	3	5	-	V	V	LN	LN	0.6	0.5	III	III	YES	YES	<1	<1	NO	NO	YES	YES	3	3
198	VIJI	24	PRIMI	35	NO	-	YES	3	3	2	-	B	B	EMER LSCS		1.7	2	I	II	YES	YES	2	5	1	1	-	-	-	-
199	KANAGA	20	PRIMI	37	NO	YES	-	-	3	3	-	V	B	LN	ABD	1.7	1.8	II	II	YES	YES	2	2	3	3	-	-	-	-
200	LEEMA	21	PRIMI	32	YES	YES	-	-	3	3	-	V	V	LN	LN	1.1	1	III	III	YES	YES	2	1	NO	NO	YES	YES	10	10

KEY TO MASTER CHART

GA	-	GESTATIONAL AGE

MATERNAL COMPLICATIONS

1	-	GESTATIONAL HYPERTENSION
2	-	PRE-ECLAMPSIA
3	-	ECLAMPSIA (IMMINENT & ANTEPARTUM)
4	-	ANEMIA (MODERATE & SEVERE)
5	-	ATONIC PPH

CHORIONICITY

1	-	DCDA (DICHORIONIC DIAMNIOTIC)
2	-	MCDA (MONOCHORIONIC DIAMNIOTIC)
3	-	MCMA (MONOCHORIONIC MONOAMNIOTIC)

PRESENTATION

V	-	VERTEX
B	-	BREECH
T	-	TRANSVERSE
C	-	COMPOUND

MOD	-	MODE OF DELIVERY

LN	-	LABOUR NATURALE
ABD	-	ASSISTED BREECH DELIVERY
O	-	OUTLET FORCEPS
LSCS	-	CAESAREAN SECTION
IPV	-	INTERNAL PODALIC VERSION

APGAR

I	-	> 8
II	-	5 – 7

III - 1 – 4

IV - 0

DIAGNOSIS AT DISCHARGE AND CAUSE OF DEATH

1 - RDS (RESPIRATORY DISTRESS SYNDROME)

2 - SEPSIS

3 - IUGR

4 - NNH (NEONATAL HYPER BILIRUBINEMIA)

5 - PDA (PATENT DUCTUS ARTERIOSUS)

6 - HYPOGLYCEMIA

7 - BIRTH ASPHYXIA

8 - ANOMALOUS BABY

9 - CORD PROLAPSE

10 - PREMATURITY AND ITS COMPLICATIONS

11 - TTTS (TWIN-TWIN TRANSFUSION SYNDROME)

12 - NNS (NEONATAL SEIZURES)

13 - IUD OF 1st TWIN